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# Adaptation to heat: differences induced isothermal strain accompanying exercise in hot and neutral environments

Jodie Maree Regan
*University of Wollongong*

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Jodie Maree Regan, B.App.Sc.



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## Abstract

Acclimatisation induces physiological changes which reduce thermal strain and may occur in response to artificial heat exposure (acclimation), and endurance exercise which elevates body core temperature. This project sought to evaluate the importance of elevated skin temperature in the thermal environment during the acclimation process, using an isothermal strain technique which kept core temperature constant between conditions. Two groups of seven healthy males, matched for peak aerobic power ($\dot{V}_{O2peak}$) and sum of six skinfolds, participated (1 hr per day for 10 consecutive days) in one of two conditions: (i) neutral physical training (NEUT: 22.4°C {s.d. 0.7}, RH 41.0% {s.d. 0.9}) or (ii) combined physical training and heat acclimation (HEAT: 38.2°C {s.d. 0.7}, RH 39.7% {s.d. 1.3}). Isothermal strain was induced by rapidly elevating rectal temperature ($T_{re}$) to 1°C above its initial resting level (cycling), then holding it constant by manipulating the external work. Subjects completed heat stress tests (39.8°C {s.d. 0.1}, RH 38.6% {s.d. 1.2}), before and after acclimation, consisting of: 20 min seated rest, 20 min cycling at 30% $\dot{V}_{O2peak}$, and 20 min cycling at 45% $\dot{V}_{O2peak}$. On the basis of pre- versus post-acclimation differences in $T_{ac}$, forehead $\dot{m}_s$ and SkBF, and perceived exertion, the HEAT condition elicited greater improvements in acclimation state, even though central thermal strain was equated between conditions. While the elevation in body core temperature is critical to acclimation, it appears necessary to expose subjects to an exogenous external thermal stress. This verifies the importance of peripheral thermoreceptors in the acclimation process, although this observation has not been demonstrated previously under conditions of isothermal strain.

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# CHAPTER ONE: INTRODUCTION

## 1.1 INTRODUCTION

Central body temperature is regulated through the balance between heat accumulation (metabolic production and environmental heat loading) and heat dissipation. Excessive heat accumulation, or a reduced ability to dissipate body heat, results in a nett body heat storage; the storage of 60 kcal will elevate the central body temperature of an average individual by 1°C (Nadel, 1979) . In comparison with the broad temperature range for terrestrial climates, central body temperature in humans, is usually regulated within a very narrow range (35° to 41°C), within which physiological function is optimal. Consequently, both behavioural and homeostatic mechanisms have evolved to defend body core temperature.

Behavioural modifications help humans better tolerate heat stress. For example, the use of thermal protective clothing, limiting exposure to heat sources, reducing the physical activity during heat exposure, and increasing rest to work ratios when conditions are extremely hot. Through physiological temperature regulation, humans are also able to defend central body temperature. Heat is actively transported from the core to the periphery, via the circulatory system. Once at the periphery, vasodilation of cutaneous blood vessels enables heat carrying blood to be exposed to a large surface area. Evaporation of eccrine sweat cools the skin surface, facilitating heat flux from capillaries to the skin surface, and then to the environment.

However, body temperature regulation may be inadequate under extremely hot conditions, particularly during prolonged exercise. The combination of prolonged exercise and heat exposure forces the cardiovascular system to provide blood flow to exercising skeletal muscle to satisfy metabolic demands, and blood flow to the skin to dissipate the heat released from the exercising muscle (Rowell, 1974). Most people can meet this dual demand, at least for a short duration. However, during prolonged exercise in the heat, even highly trained individuals may fail to maintain thermal

homeostasis, and will move into positive heat storage. In such situations, cardiac output is exceeded by the combined demands for skin and muscle blood flow (Kenney and Johnson, 1992). When this occurs, blood pressure declines, as the volume of the dilated capacitance vessels exceeds the circulating blood volume. The maintenance of blood pressure becomes the most important factor in the heat stressed individual (Rowell, 1974), and cutaneous vasoconstriction prevents pooling of blood in dependent cutaneous veins. Once this point is reached, heat dissipation is compromised, and continued exercise will rapidly elevate core temperature, eventually leading to physiological dysfunction.

Over time, humans have evolved so that heat dissipation and conservation mechanisms are able to adapt to a range of environmental conditions, as well as to the combined thermal stressors of exercise and external heat. Perhaps the most striking physiological effects for improving heat tolerance may be attributed to acclimatisation. Acclimatisation describes the physiological changes induced through natural climatic conditions, which reduce the strain of an organism. Acclimation describes the process wherein such changes occur in response to artificial manipulation of the environment (Bligh and Johnson, 1973).

As a result of natural acclimatisation, Europeans living in tropical climates display similar physiological responses to heat stress as those who acquire such responses through an artificial acclimation protocol. Typically, these adaptations include: lower core and skin temperatures, a lower heart rate, and a higher sweat rate than unacclimated controls (Hellon *et al.*, 1956). The Australian Aborigines living in traditional communities also show evidence of natural acclimatisation, by producing higher rates of sweating than their European counterparts, with rapid sweat suppression in a humid environment (Macfarlane, 1973).

The reduced physiological strain following acclimatisation is mediated by several factors. For example: an expanded plasma volume resulting from reduced plasma protein loss to the interstitial fluid (Senay, 1979), causing stroke volume to increase; a lower heart rate, which seems to be a reflex response to increased stroke volume, and

lowered skin and core temperatures; and an enhanced (sudomotor) sweating response, through increased sweat sensitivity in relation to core temperature, and a decreased core temperature threshold for sweating (Nadel *et al.*, 1974). Such adaptations minimise the effects of thermal stress upon the internal environment, permitting reduced strain and elevated tolerance, both at rest and during exercise.

Since both passive heat exposure and thermoneutral exercise elevate core temperature and induce thermal strain, the possibility exists for acclimation to be induced simply through exercise stress. Such an approach has been shown to be effective (Gisolfi and Robinson, 1969; Henane *et al.*, 1977; Pandolf *et al.*, 1977), but little is known about the mechanism underlying this process. Intuitively, one would assume that such an adaptation would depend upon an elevated body temperature. However, evidence from previous research provides little insight into the nature of this stimulus. We do not know whether it is sufficient to simply elevate body core temperature to induce exercise related acclimatisation (thermoneutral exercise), or whether it is essential to increase both core and peripheral temperature (exercise in the heat). Since we know that both core and peripheral temperatures provide critical input to the thermoregulatory process, one would assume that a more complete adaptation would be derived from the combination of endogenous and exogenous thermal stress. In this project, an attempt was made to differentiate between the role of core and skin temperature using an isothermal strain technique, in which body core temperature was elevated and maintained constant across two different environments.

## 1.2 AIMS AND HYPOTHESES

It was the purpose of this project to investigate changes in the control of sudomotor and cutaneous vasomotor responses to physical training in a neutral environment, and to combined training and heat acclimation protocols. The presence of an acclimation effect was defined as a significant decrease in auditory canal temperature ($T_{ac}$). Both protocols were designed to induce isothermal strain by a 1°C elevation in central body temperature; to maintain this elevation for equivalent duration; and to repeat the exposure for ten consecutive days.

It is hypothesised that:

(i) physical training in a neutral environment, and combined physical training and heat acclimation will both produce an equivalent lowering of the body core temperature thresholds for the sudomotor response to a standard thermal stress;

(ii) physical training in a neutral environment, and combined physical training and heat acclimation will both produce equivalent elevations in the sudomotor response sensitivities to a standard thermal stress;

(iii) sudomotor and cutaneous vasomotor steady-state responses to a standard thermal stress will be greater following combined physical training and heat acclimation than observed following neutral physical training.

# CHAPTER TWO: LITERATURE REVIEW

## 2.1 INTRODUCTION

A feeling of increased fatigue during exercise is a common experience at the beginning of a period of hot weather. However, a physiological adaptation process takes place during the ensuing weeks, diminishing fatigue and improving the ability for sustained exercise. These physiological changes can be brought about in response to natural climatic changes (acclimatisation: Hellon *et al.*, 1956), artificial heat exposure (acclimation: Nadel *et al.*,1974; Roberts *et al.*,1977, Wells *et al.*, 1980) and to endurance exercise producing significant elevations in body core temperature (Gisolfi and Robinson,1969; Henane *et al.*,1977; Pandolf *et al.*,1977). This project shall investigate changes in sudomotor and cutaneous vasomotor responses to heat stress following both acclimation and endurance training.

Senay *et al.* (1976) described the acclimation process in terms of three general phases. The initial exposure induces vasomotor and sudomotor responses resulting in reduced venous return, cardiac filling pressure and stroke volume. In compensation, cardiac frequency ($f_c$) increases to maintain blood pressure and cardiac output. Next, blood volume increases, which increases venous return, cardiac filling pressure, and stroke volume; and body core and skin temperatures are reduced as a result of increased sweat rate ($\dot{m}_{sw}$) and evaporation. Finally, body core and skin temperatures attain a new steady state level, achieved through increased sudomotor capacity and sensitivity (gain), resulting in decreased cutaneous vasodilation.

### 2.1.1 Sudomotor control and adaptation to heat stress

The body produces heat via several avenues including: basal cellular metabolism, muscular activity, and the effects of temperature, epinephrine and thyroxine on cells. To maintain thermal homeostasis, heat production must be balanced by heat loss mechanisms: radiation, conduction, convection, and evaporation. However, during

exercise in the heat, metabolic heat production can outweigh the thermoregulatory responses for heat dissipation, and core temperature ($T_c$) increases. To minimise this increase in $T_c$, there is greater dependency upon evaporative heat loss, through the secretion of sweat onto the skin surface by the eccrine glands, allowing evaporative cooling in the conversion from liquid to water vapour. An increase in $\dot{m}_{sw}$ is one of the most pronounced effects of acclimation to heat.

The eccrine sweat glands are primarily controlled by the thermoregulatory center(s). The sudomotor signals descend from the preoptic anterior hypothalamus to the eccrine gland through innervation of post-ganglionic sympathetic cholinergic fibers. Such signals reach the glands in waves, resulting in pulsatile sweat secretion. The periodicity of these neural impulses causes alternating periods of high and low peaks of sweating activity, making sweating a cyclic process (Randall, 1946b). The increase in thermoregulatory sweating closely parallels the increase in body temperature and as the sweating increases towards maximal levels, there is a recruitment of progressively more sweat glands. Initially, glands are recruited at the more distal regions, and after total recruitment there is an increase in sweat secretion per gland (Hertzman *et al.*, 1952; Randall, 1946a).

The primary input to the thermoregulatory controller is provided by $T_c$, with skin temperature playing a smaller role. Wyndham (1965) studied the relative importance of $T_c$ to mean skin temperature ($\overline{T}_{sk}$) and reported that in an ambient temperature ($T_A$) of 36°C, a 1°C rise in rectal temperature ($T_{re}$) had 4.5 times greater impact than the same rise in $\overline{T}_{sk}$. Nadel *et al.* (1971a) independently varied $T_c$, $\overline{T}_{sk}$, and local skin temperature ($T_{skl}$) to determine the importance of skin temperature in the regulation of sweating. The relationship between $T_c$ and $\overline{T}_{sk}$ in the control of local sweating rate, was found to be a summation of temperature signals in the determination of efferent activity. The $T_{skl}$ contribution was identified as a modifying effect upon the output from the central controller, acting as a multiplier in the determination of local sweating rate.

Acclimation to heat is characterised by an increase in $\dot{m}_{sw}$ (Libert *et al.*, 1983;

Sato *et al.*, 1990), however, the mechanisms underlying this response are unclear. For example, the extent to which the response is of central origin (threshold change), depicted by an earlier appearance of sweat, or of peripheral origin (change in sensitivity), where more sweat is produced for a given change in $T_c$, is uncertain. While the changes in threshold can be considered as central in origin, the precise site of modification has not been isolated (Nadel, 1979). The changes in the sensitivity of the sweating response are usually the result of modifications to the autonomic nervous system, such as heightened glandular activity per unit of neurotransmitter released (Nadel, 1979).

Evidence suggests that peripheral adaptation to repeated heat exposure may be the underlying mechanism for increased $\dot{m}_{sw}$. This has been shown in studies where greater sweating responses were elicited by pharmacological stimulation. Collins *et al.* (1965) reported that repeated daily injections of methacholine increased the local sweating response in a manner similar to that observed with acclimation. Sato and Sato (1983) found that subjects who sweated heavily had greater eccrine gland volumes, larger tubular diameters, and longer secretory tubules than subjects with poor sweating characteristics. The effects of heat acclimation on these sweat gland characteristics showed that nine months of continuous heat exposure increased tubular length and volume, and the secretory capacity of the glands (Sato *et al.*, 1990). Fox *et al.* (1964) investigated the effects of repeated local forearm heating (immersion) on the local sweating response, while acclimatising subjects using a controlled hyperthermia technique that passively elevated $T_c$. Total body $\dot{m}_{sw}$ showed large increases in response to the controlled hyperthermia sessions. Repeated local heating of the forearm during the acclimation sessions, dramatically increased $\dot{m}_{sw}$ at this site, leading to the conclusion that increased $T_{skl}$ increased the local secretory capacity at the forearm, resulting from repeated heat exposure.

Thermoregulatory function and heat tolerance may be improved as a result of exercise training in a neutral environment, which elevates $T_c$ sufficiently. In the study by Nadel *et al.*(1974), 10 days of cycle exercise at 70-80% of peak oxygen consumption ($\dot{V}_{O2peak}$) produced a large increase in $\dot{m}_{sw}$ sensitivity. Similar changes

were found by Avellini *et al.*(1982) where sweat sensitivity increased following land training 5 days a week for 20 days at 75% $\dot{V}_{O2peak}$. While these studies suggest a peripheral adaptation, through increased sweating sensitivity, exercise has been shown to produce central adaptations. Henane *et al.* (1977) showed that three months of interval training, alternating cycle exercise at 25% and 125% $\dot{V}_{O2peak}$, for 30 and 60 seconds respectively, until exhaustion, lowered the $T_c$ threshold for sweating. The important aspect of training in a cool environment is the maintenance of elevated $T_c$ for optimal acclimation results (Avellini *et al.*, 1982; Nadel *et al.*, 1974).

Acclimation is typically induced through regular exercise in the heat. Nadel *et al.* (1974) studied six subjects during physical training, and then in a combined exercise and heat acclimation protocol. They found that physical training increased the sensitivity (gain) of the sweating response (resistance hygrometry) per unit change in $T_c$, while physical training in the heat resulted in further enhancement of the sweating response, via a lowering of the threshold for the central nervous system drive for sweating. Nadel *et al.* (1974) attributed a distinct mechanism to the increase in $\dot{m}_{sw}$ during each of the two acclimation protocols, as being either central or peripheral in origin. However, had subjects not been exposed to the physical training protocol initially, both central and peripheral changes in $\dot{m}_{sw}$ may have resulted from the heat acclimation protocol studied in isolation. Shvartz *et al.* (1979) studied ten subjects in one of two acclimation regimes, and found that the sweating response following training in both temperate and hot environments was characterised by the initiation of sweating at a lower $T_{re}$. In a heat acclimation regime that controlled $T_c$ (Havenith and van Middendorp, 1986) it was found that this method of acclimation produced both central and peripheral adaptations by lowering the threshold for sweating and increasing sensitivity. This observation of both a central and peripheral adaptation has also been supported by Wells *et al.* (1980), using the whole body method of monitoring weight reductions, and it was concluded that subjects who were both heat and exercise acclimated were able to dissipate a given thermal load as a result of increased sensitivity while maintaining a lower $T_{re}$ for sweating. While it has been shown that both central and peripheral mechanisms may increase $\dot{m}_{sw}$, the attribution of a distinct mechanism following either physical training or heat acclimation has not been isolated.

When exercising in the heat, the gradient for sensible heat exchange diminishes and heat dissipation through sweat becomes vital. The increase in $\dot{m}_{sw}$ is one of the most pronounced effects of acclimation, whether it be an adaptation centrally, peripherally, or both, and since elevated $T_c$ is the crucial stimulus, adaptation may occur in response both to neutral exercise training alone and to exercise training in the heat. However, while it is known that skin temperature is important in the control of sweating, its importance in the process of acclimation is not clear.

### 2.1.2 Vasomotor control and adaptation to heat stress

While sweating serves to transfer heat from the skin to the air, skin blood flow (SkBF) is the most important mechanism for transferring heat from the core to the skin during heat stress. In situations where sweating does not occur, that is, lower core and skin temperatures, SkBF elevation brings skin temperature closer to the temperature of the blood, while lowering SkBF brings skin temperature closer to $T_A$. Thus, the body is able to modify convective and radiative heat loss by influencing skin temperature through the variation of SkBF. Cutaneous blood vessels dilate and constrict in response to neural commands from the hypothalamus. These signals are initiated in response to thermal input from both central and peripheral thermoreceptors. Increased blood flow in the acral regions (feet, hands, lips, nose, and ears) occurs by sympathetic nervous tone withdrawral (Gaskell, 1956). These regions are solely innervated by adrenergic sympathetic nerves (Johnson, 1986) which regulate constrictor tone. In non-acral skin areas the increase is obtained by active vasodilation (Blair *et al.*, 1961). Wenger *et al.*(1985) demonstrated active dilation of blood vessels by heating (39°C) or cooling (27°C) forearm skin and comparing the response to a control forearm (33°C). It was found that local heating of the forearm caused a vasodilatory effect in the heated forearm.

During exercise in the heat, the primary task of the cardiovascular system is to simultaneously provide blood flow to the exercising skeletal muscle, and sufficient SkBF to dissipate the heat released from the exercising muscle. In hot environments,

increased skin temperature produces a relatively narrow core-to-skin thermal gradient. In response to the high skin temperature, SkBF increases to achieve sufficient heat transfer for thermal balance (Roberts and Wenger, 1979). Even though heat transfer to the environment may be enhanced by dilation of the cutaneous veins, circulatory strain may be imposed through an accumulation of blood in the vessels of the skin. This causes a displacement of blood from the thorax which may reduce central venous pressure and thus cardiac filling pressure (Rowell, 1983). In compensation, reflex vasoconstriction in the splanchnic and renal regions occurs (Rowell, 1974) so that cardiac output may be diverted to the skin and exercising muscle.

Exercise creates a thermoregulatory drive for cutaneous vasodilation (Johnson and Rowell, 1975), as well as a non-thermoregulatory drive for vasoconstriction (Hirata *et al.*, 1983). The latter suggests several distinct reflex effects including initial vasoconstriction with the onset of dynamic exercise (Hirata *et al.*, 1983), elevated $T_c$ threshold for vasodilation during exercise (Rowell, 1974), and an attenuated rise in SkBF at high core temperatures during prolonged exercise (Brengelmann *et al.*, 1977). The initial vasoconstriction and elevated $T_c$ threshold for vasodilation result in the heat loss early in exercise being much less than the heat produced by muscular contraction. Consequently, the majority of the heat produced during the first few minutes of exercise is stored causing $T_c$ to rise. A sufficient elevation in $T_c$ beyond the threshold for vasodilation then permits heat dissipation, through rising SkBF, in response to elevated metabolic heat production (Rowell, 1974). However, above a $T_c$ of approximately 38°C an attenuated rate of increasing SkBF occurs (Brengelman *et al.*, 1977). Nadel *et al.* (1979) suggested this attenuation may arise from a decreased central filling pressure, since a plateau in the SkBF response to rising $T_c$ did not occur during supine exercise.

The vasomotor response to heat stress may not always be advantageous to thermoregulation, as in high intensity exercise which causes constriction, and prolonged exercise causing blood pooling and decreased cardiac output. Therefore, initial plasma volume changes are important to the process of heat acclimation. Senay *et al.*(1976) attributed the attainment of cardiovascular stability to the expansion of plasma volume during the first four days of heat exposure in an acclimation regime. It was suggested

that during the initial phase of heat exposure, elevated $f_c$ and reduced stroke volume in the presence of dilated cutaneous blood vessels, allowed proteins to be flushed into the vascular volume via the lymph ducts, causing plasma volume to expand (Senay *et al.*, 1976).

The SkBF response to different acclimation regimes is variable in the literature. In a study where the elevation in SkBF was induced by passive elevation of body temperature (Fox *et al.*1963), peripheral blood flow was higher for a given body temperature by the end of heat acclimation. Roberts *et al.* (1977) further studied the relationship of $T_c$ to SkBF and found that SkBF was higher at a given $T_c$ following both exercise training and heat acclimation. A slight reduction in forearm SkBF was reported by Havenith and van Middendorp (1986) following 7 days of heat acclimation that controlled $T_c$ during exposures.

Previous acclimation experiments studying changes in SkBF are generalised from forearm blood flow measurements, using venous occlusion plethysmograpy. Despite the demonstrated reliability of this technique (Longhurst *et al.*, 1974; Saumet *et al.*,1986), certain methodological limitations exist. First, measurements can only be taken from the distal regions of the limbs, and may not generalise well to other body regions. For example, Havenith and van Middendorp (1986) found forearm blood flow to be reduced following acclimation and suggested that since the relationship between core and skin temperature remained equal with acclimation, SkBF in other parts of the body may not have been decreased. Nishiyasu *et al.* (1992), in a comparison study of the blood flow response of the forearm and calf to thermal stress during dynamic exercise, found blood flow differences between the two regions, and suggested that the SkBF response may not be uniform over the entire body surface. Second, the ability to follow rapid changes is limited because the method is discontinuous, and measurement frequency of forearm blood flow is restricted to two to four times per minute. Third, the method measures total forearm blood flow and does not differentiate between skin and muscle blood flow (Johnson *et al.*, 1984). Finally, since venous occlusion is necessary for every measurement, it is unknown to what extent the technique interferes with blood flow, producing a reactive error.

A more recently developed method, laser Doppler velocimetry, is dependent on the Doppler shift of laser light reflected from moving particles (Johnson *et al.*, 1984). This method has several advantages over plethysmography including: its application to multiple skin sites, its minimal reactive error, a continuous signal output, its rapid response time, and its specificity to surface tissues less than 1.5mm in depth (Johnson *et al.*, 1984). This technique shall be employed to measure SkBF in the present study.

### 2.1.3. Acclimation to endogenous and exogenous heat stress

During acclimation, the thermal strain associated with endogenous or exogenous heat stress is gradually reduced. People experience decreased cardiac frequency, core temperature, and skin temperature, and increased sweat rate (Nadel *et al.*, 1974; Pandolf, 1979; Rowell,1974). These improvements in thermoregulatory function may arise following physical training in a neutral environment or exercise in the heat (Mitchell *et al.*, 1976; Pandolf *et al.*, 1977; Shvartz *et al.*, 1979).

Fox (1968) suggested that an increase in $T_c$ formed the prime drive for the activation of thermoregulatory adaptations. However, Chen and Elizondo (1974), through investigations of the differences between whole body and $T_{skl}$ effects on acclimation, concluded that an increased central stimulus ($T_c$), in conjunction with increased $T_{skl}$ (heat) were needed for acclimation to heat.

Extended duration dynamic exercise increases metabolic heat production, and when such heat production exceeds heat dissipation, the exercise causes a rise in $T_c$, which eventually reaches the thermoregulatory thresholds for cutaneous vasodilation and sweating (Johnson, 1986). Gisolfi and Cohen (1979) identified the critical factors involved in this interaction between heat production and $T_c$ elevation as the environmental conditions, and the duration and intensity of the exercise. Physical training programmes which lead to a prolonged increase in $T_c$ prove beneficial to the acclimation process. Most researchers agree that physical training improves physiological responses during exercise in the heat and hastens the process of heat

acclimation (Nadel *et al.*, 1974; Pandolf *et al.*, 1977; Roberts *et al.*, 1977). The general consensus for optimal results from physical training include: strenuous interval training or continuous exercise at an intensity greater than 50% $\dot{V}_{O2peak}$ (Henane *et al.*, 1977; Pandolf *et al.*, 1979). Gisolfi and Cohen (1979) suggest that this type of training may approximate 50% of the improvements gained by combined exercise and heat acclimation.

The effect of physical training on the acclimatisation process has been found to be $T_c$ specific. Avellini *et al.* (1982) compared land training and training in two different water temperatures (20°C and 32°C) over a four week period. $T_{re}$ was monitored during training periods and increased 1.1°C and 0.6°C for the land and 32°C water-trained groups respectively. However, during the 20°C water-training protocol, $T_{re}$ decreased. Following the four week exposure, it was found that heat tolerance improved for the land and warm (32°C) water-trained subjects, and it was suggested that this was the result of the greater elevations in $T_c$ during the exercise sessions. Hessemer *et al.* (1986) used a "sweatless exercise" protocol, whereby the $f_c$ of each subject was kept at a constant 120 $b.min^{-1}$, while maintaining an approximately constant mean body temperature ($\overline{T}b=0.87T_c+0.13\overline{T}_{sk}$). Sweating was suppressed by adjusting $T_A$, to decrease $\overline{T}_{sk}$, in compensation for the exercise-induced elevation of $T_c$. It was found that training which was not associated with a $T_c$ elevation, did not change the threshold for sweating onset, or the sensitivity of the sweating response in relation to mean body temperature. Hessemer *et al.* (1986) concluded that the threshold alterations found in athletes who trained on land suggest the thermoregulatory characteristics are a result of adaptation to the internal heat load accompanying endurance exercise.

While it is known that the body can adapt to repeated thermal stress, both of endogenous and exogenous origin, we do not have a full understanding of differences between acclimation states induced by these means. Previous groups have compared acclimation from both sources of heat stress, but few groups have tried to equate physiological strain between conditions. Failure to allow for this renders acclimation differences difficult to interpret. Many studies have exposed subjects to a constant exogenous heat stress. This was an inefficient method of acclimation since there is a

progressive decline in the stimulus to further adaptation due to the increase in efficiency (Fox, 1968). This method for studying heat acclimation also requires that the stimulus to heat adaptation be measured and expressed in terms of all the variables contributing to the heat load including: $T_A$, relative humidity, air velocity, radiation, and clothing. Since investigators have used different levels of these variables, comparing the results of one study to another is difficult (Fox, 1968).

Various studies have attempted to equate strain in acclimation regimes using $f_c$ as the prime regulator. This method involves subjects working at a $f_c$ that corresponds to a certain percentage of their $\dot{V}_{O2peak}$. However, since decreased $f_c$ is one of the first adaptations to exercising in the heat, a constant stimulus is no longer present to produce further adaptation, causing the method to be inefficient. In some studies (Nadel *et al.*, 1974; Roberts *et al.*, 1977; Avellini *et al.*, 1982), the exercise intensity was increased to maintain $f_c$ within a prescribed range as subjects increased their fitness on successive days. As a result of heat loading elevating $f_c$, the exercise intensities differed between neutral and heat protocols. Since $f_c$ responds to variables in addition to thermal strain, it is possible that this method failed to equate thermal strain between groups. Logically then, one must use a variable which primarily reflects thermal load to equate strain.

With the rise in $T_c$ being the primary drive for acclimation, it seems logical that projects investigating the relative roles of $T_c$ and $\overline{T}_{sk}$ in the acclimation process, would seek to control one or more of the independent variables stimulating thermal adaptation. Such isothermal (controlled hyperthermia) paradigms are rarely found within the literature, resulting in an incomplete understanding of the stimuli for heat acclimation. There are, however, two exceptions. Fox (1968) controlled $T_c$ by maintaining it constant at an elevated level, by means of an increasing $T_A$. It was shown that a protocol of passive exposure to heat, at a constantly elevated $T_c$, was a very efficient method of acclimation. Havenith and van Middendorp (1986) revised this regime, utilising a work to rest protocol in the heat once $T_c$ had been elevated to a particular level, thus inducing constant heat strain. These studies indicate that acclimation occurs as a result of controlling $T_c$. By controlling this variable then, it may be possible to determine the importance of $\overline{T}_{sk}$ during the acclimation process. While these projects

were not designed to evaluate the roles of central and peripheral stimuli in the acclimation process, the methods adopted provide a valuable model for such an assessment. Consequently, their method of elevating and maintaining a constant $T_c$ has been adopted in this project.

### 2.1.4 Summary

At this point it is known that both artificial acclimation and physical training generally improve heat tolerance through changes occurring either within the central control, or the peripheral responses to thermal efferents. However, gaps exist within our knowledge. Most studies looking at the differential role of training and acclimation have failed to equate the central body thermal strain across protocols. A clearer discrimination needs to be made between the influence of physical training and acclimation during this testing. Little is known about the steady state responses to thermal stress following acclimation, that is, when both sudomotor and cutaneous vasomotor responses have saturated. Although both Nadel *et al.* (1974) and Roberts *et al.* (1977) found enhanced sweating responses, their protocols dealt with exercise transients rather than investigating the heat exchange processes during steady state conditions found in most thermoregulatory studies (Havenith, 1985). Since heat tolerance is not merely a function of the response dynamics, but is related to steady-state function, an understanding of this feature is essential to better understand the physiology of acclimation. Furthermore, little is known about the differences in the thermal responses of different skin regions. For example, it is not known whether previous observations are region specific or well generalised. This is especially relevant to SkBF, due to site limitations of previous measuring techniques.

Research demonstrates that thermal strain may be reduced as a result of acclimatisation in the natural environment, in response to exercise in neutral conditions which sufficiently elevate $T_c$, and following exercise in the heat. However, this research is inconclusive as to the mechanisms which cause such physiological adaptations. Since body temperature regulation may be inadequate in hot conditions, and particularly during prolonged exercise, the study of the acclimation process is

important to begin to understand the control of such adaptations. This project attempts to determine the importance of skin temperature in optimising heat adaptations, using an isothermal strain technique which equates body $T_c$ across two different environmental states.

# CHAPTER THREE: METHODS

## 3.1 METHODS

### 3.1.1 SUBJECTS

Two groups of healthy, active males, matched for aerobic power (peak oxygen consumption: $\dot{V}_{O2peak}$; Q-Plex I[1]) and sum of six skinfolds (Σ6skf), participated in one of two exercise conditions (Table 3.1): physical training ($T_A$ 22.4°C) or combined physical training and heat acclimation ($T_A$ 38.2°C). Each subject received a Subject Information Package, provided informed consent, completed an activity questionnaire, and satisfactorily completed the Physical Activity Readiness Questionnaire (PAR-Q, 1962) before participating.

Acclimation was induced by training both groups for one hour per day for ten consecutive days, while maintaining a constant and equivalent body $T_c$ between conditions. Isothermal strain was achieved in each condition by rapidly elevating $T_{re}$ to 1°C above its initial resting level through cycling, and holding that elevation by manipulating the rate of external work within each environment.

### 3.1.2 PROCEDURES

#### 3.1.2.1 Subject matching

Following measurement for mass and height, the sum of skinfolds was determined from measurements taken from six sites: biceps (vertical fold at the mid-acromiale-radiale line on the anterior surface of the arm); triceps (vertical fold at the mid-acromiale-radiale line on the posterior surface of the arm); subscapular (fold beneath the inferior angle of the inferior angle of the scapula at an oblique angle of 45°

[1] Details of apparatus are provided in section 3.1.3.

**Table 3.1:** Characteristics of subjects.

| Subject | Age (yrs) | Mass (kg) | Height (cm) | Σ6skf (mm) | $\dot{V}_{O2peak}$ | $\dot{W}_{peak}$ (watts) |
|---|---|---|---|---|---|---|
| NEUT 1 | 22 | 81.9 | 175.5 | 77.6 | 54.1 | 396 |
| 2 | 20 | 73.6 | 179.6 | 84.5 | 71.0 | 360 |
| 3 | 20 | 69.3 | 178.0 | 87.0 | 66.5 | 360 |
| 4 | 21 | 81.2 | 176.9 | 106.4 | 57.3 | 376 |
| 5 | 19 | 69.9 | 180.2 | 87.0 | 62.6 | 396 |
| 6 | 18 | 80.7 | 185.7 | 84.5 | 55.6 | 360 |
| 7 | 18 | 89.1 | 179.4 | 113.0 | 46.8 | 324 |
| mean | 19.7 | 78.0 | 179.3 | 91.4 | 59.1 | 367.4 |
| s.d. | 1.5 | 7.3 | 3.3 | 13.0 | 8.2 | 25.0 |
| HEAT 1 | 26 | 76.1 | 166.0 | 72.0 | 52.6 | 306 |
| 2 | 20 | 66.7 | 176.5 | 39.3 | 57.1 | 321 |
| 3 | 23 | 60.1 | 176.4 | 44.5 | 73.7 | 378 |
| 4 | 18 | 83.8 | 179.4 | 98.6 | 53.2 | 368 |
| 5 | 19 | 81.7 | 182.9 | 50.4 | -- | 380 |
| 6 | 21 | 84.0 | 180.3 | 96.5 | 48.1 | 324 |
| 7 | 26 | 62.8 | 168.9 | 68.9 | 67.8 | 348 |
| mean | 21.9 | 73.6 | 175.8 | 68.1 | 58.8 | 346.4 |
| s.d. | 3.2 | 10.3 | 6.2 | 23.3 | 9.9 | 29.9 |

Abbreviations: NEUT = neutral physical training; HEAT = combined physical training and heat acclimation; Σ6skf (sum of six skinfolds); $\dot{V}_{O2peak}$ (peak oxygen consumption in $ml.kg^{-1}.min^{-1}$); $\dot{W}_{peak}$ (peak work rate); -- (data not obtained)

running downwards from the horizontal; axilla (vertical fold on the mid axillary line at the level of the xiphoid process with the right hand placed on the head); suprailiac (oblique fold running at an angle 45° downward from the anterior superior iliac spine); abdominal (vertical fold which is raised 5 cm lateral to, and at the level of the omphalion, supraspinale (fold 7 cm above the spinale on the line to the anterior axillary border)) (Modified after Ross *et al.*, 1982). All measurements were taken on the right side of the body.

The $\dot{V}_{O2peak}$ of each subject was determined using a ramp cycle protocol, with work rate increments of 3 watts every 5 seconds, beginning at 0 watts ($T_A$ of 22°C). Each test was terminated at volitional fatigue, with a reduced work rate to allow recovery. Verbal encouragement was given to each subject. Breath-by-breath data were averaged over 15 second intervals, with the highest value obtained during the final period of testing being taken as the $\dot{V}_{O2peak}$. This method was found to be the most suitable determination of $\dot{V}_{O2peak}$ (Regan and Bube, 1992; Appendix A). The $\dot{V}_{O2peak}$ test data allowed determination of the subsequent step work rates used later in the heat stress tests (30% and 45% peak watts attained at $\dot{V}_{O2peak}$). $f_c$ was monitored using a SportTester, and peak $f_c$ was taken as the highest value obtained during the test.

Subjects were then matched so that significant group differences did not exist between the variables: mass, $\dot{V}_{O2peak}$, and Σ6skf.

### 3.1.2.2 Heat stress test

Subjects were exposed to two heat stress tests: one preceding, and one following acclimation. Each test was conducted in a hot, dry climate ($T_A$ 39.8°C ± 0.1, relative humidity 38.6 ± 1.2). Subjects first rested and then cycled at two different relative intensities: (i) rest (20 minutes); (ii) 30% work rate at $\dot{V}_{O2peak}$ ($\dot{W}_{peak}$) (20 minutes); and (iii) 45% of $\dot{W}_{peak}$ (20 minutes; modified after Havenith and van Middendorp, 1990). These intensities were defined as the three phases of the heat stress test (HST). Measurements during this testing phase included: skin blood flow, sweat rate, change in mass, aural and skin temperatures, heart rate, thermal sensation, and ratings of

perceived exertion.

Skin blood flow (laser Doppler velocimetry) was measured continuously from the forearm during the first 15 minutes of each phase of the HST. During the last 5 minutes of each phase, SkBF was measured at five sites (forehead, chest, upper back, upper arm, anterior thigh) for 20 seconds in each minute. $T_{ac}$ (zero gradient aural thermometry) and $\dot{m}_{sw}$ (forehead and forearm; capacitance hygrometry) were measured at 1 Hz, while skin temperatures (thermistors) and $f_c$ (SportTester) were measured at 0.2 Hz. Prior to, and throughout the one hour exposure, at 5 minute intervals, subjects were asked to rate their thermal sensation (modified from Gagge *et al.*,1967), and after commencement of exercise at 20 minutes, their rating of perceived exertion (Borg, 1962) for the whole body, the chest and the legs (after Pandolf, 1978). Immediately following the exposure, a post-exposure mass was recorded.

### 3.1.2.3 Acclimation protocol

A pilot study was conducted to evaluate the magnitude of the rise in $T_c$ which might be encountered during a typical endurance training stimulus. Five trained runners monitored their $T_{re}$ immediately prior to and after a typical training session. Trained runners $T_c$ elevations ranged from 1.4 to 1.9°C. It was therefore determined that a 1°C was a sufficient thermal stimulus (Appendix B), and was chosen as the isothermal $T_c$ target during acclimation.

A second pilot study was conducted to estimate the initial work rate required to raise $T_c$ to 1°C above resting by 30 minutes into the one hour exposure. Five males (exclusive of the current sample) were studied in both acclimation conditions. It was found that the work rate required to elevate $T_{re}$ was influenced by body mass. Therefore, the product of 1.6 watts.kg$^{-1}$ and body mass for the HEAT group and 2.1 watts.kg$^{-1}$ and body mass for the NEUT group would achieve this target rise in $T_c$. (Appendix C).

The validity of using $T_{re}$ as an index of thermal strain during the acclimation

regimes was evaluated (Appendix F), since a greater exercise load with the legs may induce a proportionately greater increase in $T_{re}$ than hypothalamic temperature. Auditory canal temperature and $T_{re}$ were monitored in four subjects who cycled at an intensity sufficient to elevate $T_{re}$ 1°C above resting in both acclimation conditions. It was found that $T_{re}$ produced a valid indication of $T_c$ since there were no significant differences in the response patterns of the two $T_c$ indicators.

The day immediately following the first HST, paired subjects began the acclimation protocol for 10 consecutive days, for one hour per day, in one of two conditions. The neutral temperature physical training group (NEUT), cycled at an intensity sufficient to elevate and maintain $T_{re}$ 1°C above its pre-exposure resting level, in an $T_A$ of 22.4°C {s.d. 0.7} and 41.0% relative humidity {s.d. 0.9}. The combined physical training and heat acclimation group (HEAT), were exposed to an $T_A$ of 38.2°C {s.d. 0.7} and 39.7% relative humidity {s.d. 1.1}, and similarly exercised at an intensity sufficient to elevate and maintain $T_{re}$ 1°C above pre-exposure resting level. $T_{re}$ was monitored closely throughout the experiment and the work rate was adjusted accordingly. Work rate changes were recorded to determine total work performed during each session.

### 3.1.2.4 Experimental standardisation

To ensure that testing was performed with minimal influence of extraneous factors, subjects were asked to avoid exercise other than that performed in the laboratory, and to abstain from food and caffeine for one hour prior to testing: subjects were also required to hyperhydrate before bed on the days prior to testing: one liter of water after 7pm; and consumption of one liter of fluid before 9am on the day of the test. Immediately before the start of each HST and each acclimation exposure, subjects consumed 400ml of water. The study was conducted during the winter (July and August) to minimise acclimatisation effects, and testing for each subject was performed at the same time of day to cater for circadian shifts in $T_c$.

### 3.1.2.5 Subject preparation

On arrival at the laboratory, subjects were asked to void and were measured for mass. The aural thermistor was inserted to allow for its stabilisation period of 20 minutes. Skin thermistors were then attached with waterproof tape. Squares of double sided adhesive tape, with a small circular hole in the middle were placed on each of the SkBF sites to allow for subsequent attachment of the blood flow probe. A Sport Tester (Polar Electro, 3000, Finland) was also fitted. During the preparation period, instructions for the psychophysical variables were read to each subject, and a baseline recording for thermal sensation was taken. Placement of the laser probe and attachment of the sweat capsules did not occur until after the subject entered the chamber.

## 3.1.3 APPARATUS

### 3.1.3.1 Peak Oxygen Consumption ($\dot{V}_{O2peak}$)

Data for the determination of $\dot{V}_{O2peak}$ were collected on a breath-by-breath basis using a Quinton Q-Plex I system (Quinton Instrument Company, Q-Plex I, Seattle) that was comprised of a pneumotachograph (Hans Rudolph Inc., model 3813, Kansas City, U.S.A.), a zirconia oxide oxygen analyser, and an infrared absorption carbon dioxide analyser. Subjects breathed through a Hans Rudolph two-way valve (dead space 115 ml), connected to the Q-Plex I by 35mm of low resistance tubing. A headset held the valve in position while allowing freedom of movement. Subjects cycled on an electronically braked cycle ergometer (Lode Excalibur Sport, Netherlands). The seat and handlebars were adjusted in the vertical and horizontal planes for subject comfort.

Validation of oxygen ($O_2$) and carbon dioxide ($CO_2$) concentrations was performed using expired gas samples taken from a single subject who exercised on a cycle ergometer (Monark, Sweden) at work rates ranging from 0 to 300 W, resulting in $O_2$% and $CO_2$% values ranging from 15.48 to 17.72 and 2.75 to 4.22 respectively. Independent analysis of $O_2$ and $CO_2$ concentrations in the expired air samples was

simultaneously performed using an Applied Electrochemistry $O_2$ analyser (Applied Electrochemistry Inc., model S-3A, California) and a Beckman $CO_2$ analyser (Beckman Instruments Inc., model LB-2, California). During the exercise period, gas samples were drawn from a 4 mm tube situated within the Q-Plex I mixing chamber adjacent to the Q-Plex I gas sampling tube. Analysis of the comparisons between the independent analysers and the Q-Plex I resulted in correlation coefficients of 0.956 for $O_2$% and 0.972% for $CO_2$% (Figures 3.1A & 3.1B). Volume validation was performed by collecting expired gas samples in a water filled wedge spirometer (Phillip Harris Limited) connected in a series with the Q-Plex I using low resistance tubing (35 mm diameter). Tidal volumes ranging from 501 to 4842 ml were exhaled into the system as single breaths, and the volumes recorded from the spirometer converted to body temperature and pressure saturated (BTPS) conditions. Comparisons of these volumes with the corresponding tidal volume from the Q-Plex I produced a correlation coefficient of 0.999 (Figure 3.1C).

### 3.1.3.2 Skin Blood Flow

Data for SkBF was collected at 1Hz using laser Doppler velocimetry (Vasamedics Inc.,TSI Laserflo BPM$^2$, USA). This method was dependent on the Doppler shift of laser light reflected from moving particles (red blood cells; Johnson *et al.*, 1984). Collisions between photons and stationary tissue randomises the direction of the light that eventually reaches the moving erythrocytes, with the return of light to a photodetector via sensing fibers. Only photons that have been scattered by moving tissues will have undergone a Doppler frequency shift. The result is the creation of Doppler beat frequencies which generate an electrical signal at the photodetector. The signal is processed and variables including blood flow, tissue blood volume, and red blood cell velocity are computed and reported through analog outputs and a digital panel meter (Shepherd *et al.*, 1987).

The Laserflo BPM$^2$ incorporated a gallium aluminium arsenide semiconductor laser, operating in a single longitudinal mode, with a wavelength of 780 nanometers. The fiber optic probe had two receiving fibers with a core diameter of 100 microns, a numerical aperture of 0.28, and a spacing of 0.5 mm from the transmitting fiber, which

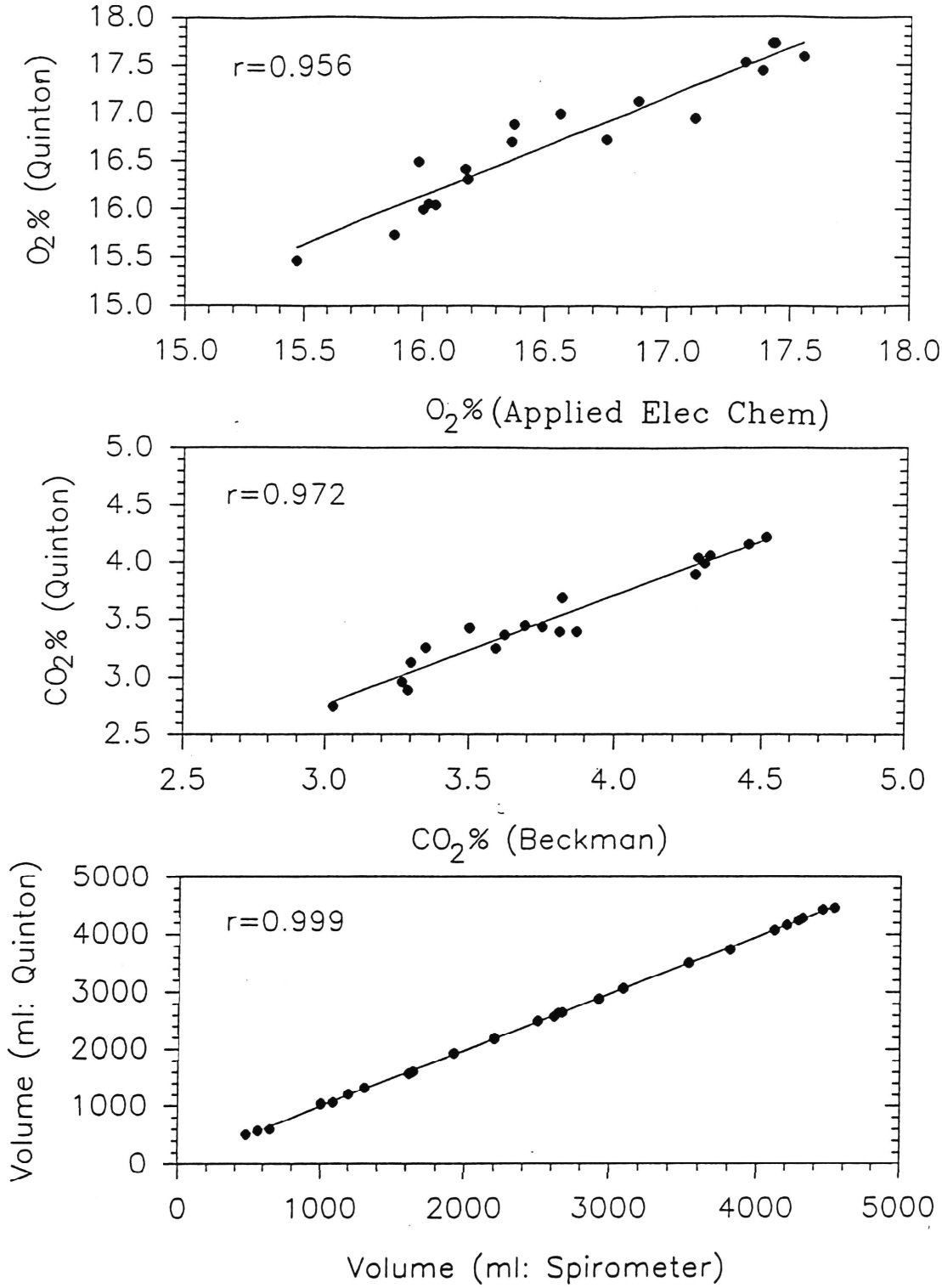


**Figure 3.1:** Validation of gas analysers and pneumotachograph output by comparisons of: (A) oxygen concentrations for Q-Plex I and Applied Electrochemistry analyser (r=0.956); (B) carbon dioxide concentrations for Q-Plex I and Beckman analyser (r=0.972); and C) expired tidal volumes for Q-Plex I and water filled spirometer (r=0.999). Data from Solomon (1991) with permission.

had a core diameter of 50 microns and a numerical aperture of 0.20 (Borgos, 1990). Prior to each test, the integrity of the fiber optic probe and the electrical zero of the system zero were checked. Analog output was passed, via an eight channel, 12-bitanalog-to-digital converter (Computer Boards Inc., PPIO-A18, Mansfield, U.S.A.), to a mains-isolated IBM compatible laptop computer (Total Peripherals, Notebook 386SX, Sydney, Australia). Data were sampled at 1 Hz.

The laser Doppler velocimeter has been validated previously by various authors using different techniques. The most conventional method, venous occlusion plethysmography, has been one method of validation (Johnson *et al.*, 1984; Saumet *et al.*, 1986; Winsor *et al.*, 1987). By incrementing SkBF through elevations in skin temperature, a near linear relationship (r=0.96), and considerable reliability was found to exist between laser Doppler velocimetry (LDV) and plethysmography. This relationship is shown in Figure 3.2A. Nilsson *et al.* (1980) utilised a fluid model to evaluate the laser Doppler flowmeter to enable the red cell velocity, concentration, and oxygen content to be individually varied and controlled in a fixed flow geometry. It was found that a linear relation existed between the flowmeter response and flux of red cells irrespective of how the product of red cell concentration and velocity was formed. *In vitro* models have been used by Shepherd *et al.* (1987), and it was observed that LDV produced stable and reproducible results and was relatively immune to fluctuations in blood oxygenation. Possibly the best method of validation was performed against the gas clearance technique (Watkins and Holloway, 1978; Kvietys *et al.*, 1985; DiResta *et al.*, 1987), which measures the removal rate of an injected gas into the skin, such as hydrogen or xenon, by cutaneous capillary blood flow. As with LDV, these measurements sample only the most superficial layers of tissue. The relationship of hydrogen clearance to LDV is (r=0.94) shown in Figure 3.2B.

#### *3.1.3.2.1 Pilot study*

Since the measurement of SkBF using point determinant methods, such as LDV, relies on the assumption that values at the measurement site are representative of the segment as a whole, a pilot study (Cotter *et al.*, 1993) was conducted to assess the

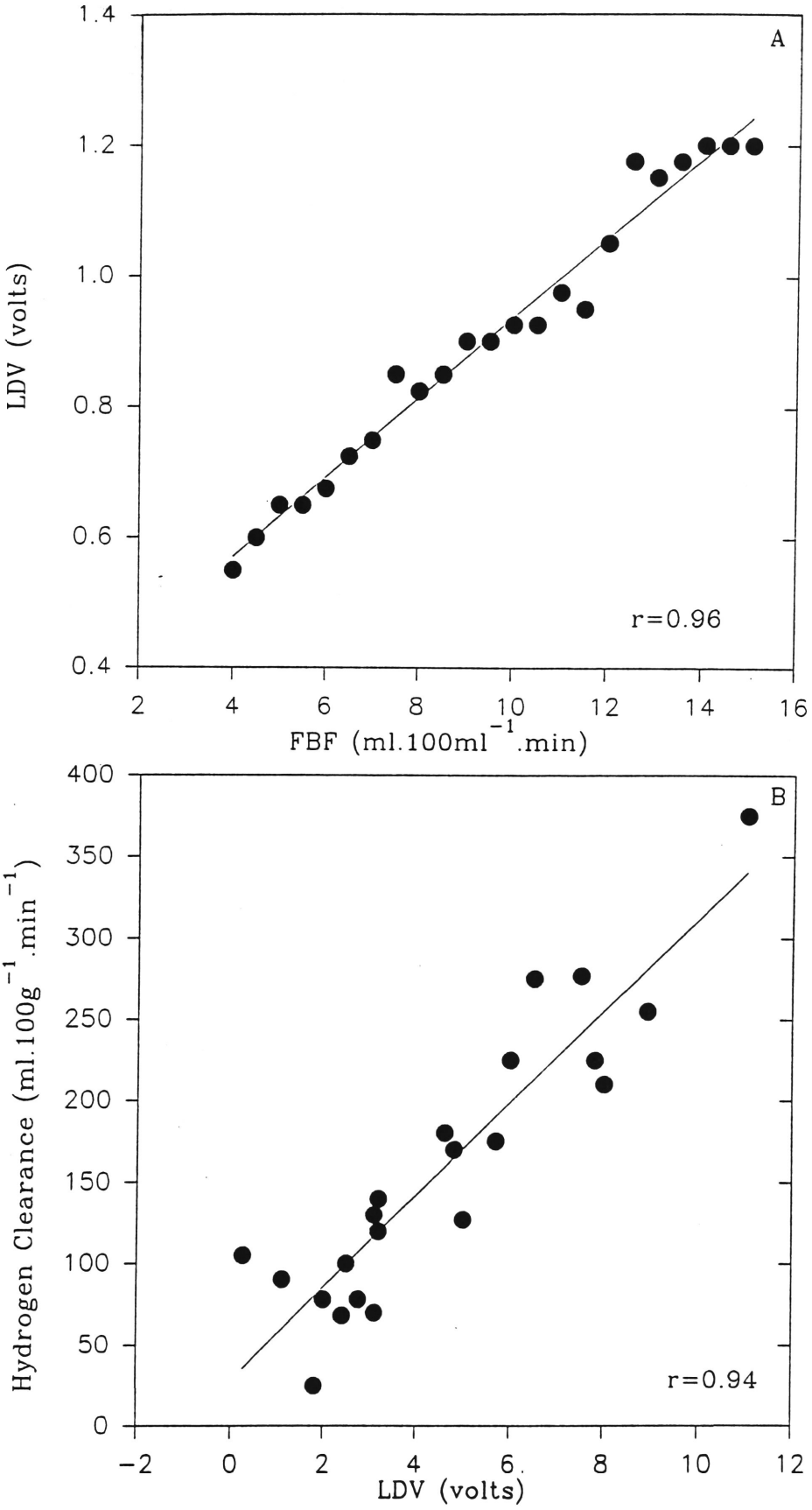


**Figure 3.2:** The relationship of laser Doppler velocimetry (LDV) to forearm blood flow (FBF) during a period of rising skin blood flow induced by elevations in skin temperature (Figure 3.2A; reproduced from Johnson *et al.*, 1984), and the relationship of LDV to tissue blood flow determined by hydrogen clearance in gastric mucosa (Figure 3.2B; reproduced from DiResta *et al.*, 1987).

uniformity of segmental SkBF, and to isolate *loci* which best reflected the average SkBF within and between body segments.

The LDV was used to obtain SkBF from six male subjects (age 27.8, s.d. 6.2 yr) during supine, thermoneutral rest ($T_A$=27°C, RH=40%). The right side of the head and right chest, back, upperarm, forearm and hand were partitioned into 16, 30, 30, 45, 60 and 44 sites respectively. SkBF, averaged at 2 Hz, was obtained for 9-sec at each site. Values exceeding 3.5 standard deviations from the mean at each site were discarded. Data for each segment were averaged to derive segmental SkBF. One-way analyses of variance were performed to examine the effect of site and segment on the mean and variability in SkBF.

The head (10.8 {s.d. 0.6} LDV units) and hand (10.2 {s.d. 3.4} LDV units) displayed higher segmental SkBF than the chest (6.0, {s.d. 2.1}), upperarm (4.6 {s.d. 0.9}), forearm (5.1 {s.d. 1.8}) and back (5.5 {s.d. 1.9}; $p<0.05$). The within-subject standard deviation in SkBF was not different between segments; 20.9 $\pm$ 11.8 % of the mean segment SkBF for the back, to 26.0 $\pm$ 12.4 % for the forearm.

Pearson's product-moment correlation was used to determine the linear relation between SkBF at each site and mean SkBF within and between segments. Sites which best correlated with segmental SkBF generally had the highest correlations between segments, with the exception of the head. The temple and mid-forehead were the best predictors of segment SkBF, whereas the lateral nose was the best head predictor of upper body SkBF. The $4^{th}$-$5^{th}$ intercostal region, immediately lateral to the sternum, had the highest correlations with mean chest SKBF ($r=0.83 \pm 0.10$). The best region of the upperarm was the medial aspect of biceps, midway between the ante-cubital fossa and axilla ($r=0.83 \pm 0.10$). A single best region was not identified for the forearm, although a group of four sites on the ventral aspect overlying the mid-point of the ulna yielded $r=0.84$ ($\pm$ 0.08). The best region for the back was over the lateral border of the scapula ($r=0.83 \pm 0.04$).

As a result of this pilot study, the following sites were selected for SkBF data

collection: forehead (right temple); chest (region between the 4th and 5th intercostal space immediately lateral to the sternum); back (upper point on the lateral border of the scapula); upper arm (medial aspect of biceps midway between the ante-cubital fossa and the axilla); forearm (midpoint of ulnar between the lateral epicondyle and styloid process on the ventral surface; thigh (midpoint of femur between head of femur and medial condyle on the ventral surface). LDV results in both relative and absolute measures of SkBF, but since the latter have not been shown to match perfectly with SkBF derived using other techniques, flow determined using LDV is typically reported as relative flows. Thus data were stored as a voltage output only. Changes in vasomotor threshold and gain could not be determined from the data due to the methods for data collection of this variable, where the probe was not continuously on the skin for the duration of the exposure, but moved between the five measurement sites.

### 3.1.3.3 Sweat Rate

Sweat rate was monitored at two sites continuously and simultaneously during the HST, at 1 Hz using a sweat capsule system (Capacitance hygrometry, Cumberland College of Health Sciences, Sydney). In this system, an air pump bubbled air at a known flow rate (400 cc.min) through a sealed flask containing a saturated salt solution (lithium chloride), which has a known standard relative humidity across the temperature range from 10-40°C (Winston and Bates, 1960). This stream of air was directed onto the skin under a sealed capsule (2.19cm$^2$). The increase in relative humidity due to the evaporation of sweat under the capsule allowed $\dot{m}_{sw}$ to be determined. Figure 3.3 shows a schematic of the sweat monitoring system. The sweat capsules were mounted on the skin at two locations, the forehead and the chest, in conjunction with previous research (Nadel *et al.*, 1974; Roberts *et al.*, 1977). Analog output was passed, via an eight channel, 12-bit analog-to-digital converter (Computer Boards Inc., PPIO-A18, Mansfield, U.S.A.), to a mains-isolated IBM compatible laptop computer (Total Peripherals, Notebook 386SX, Sydney, Australia).

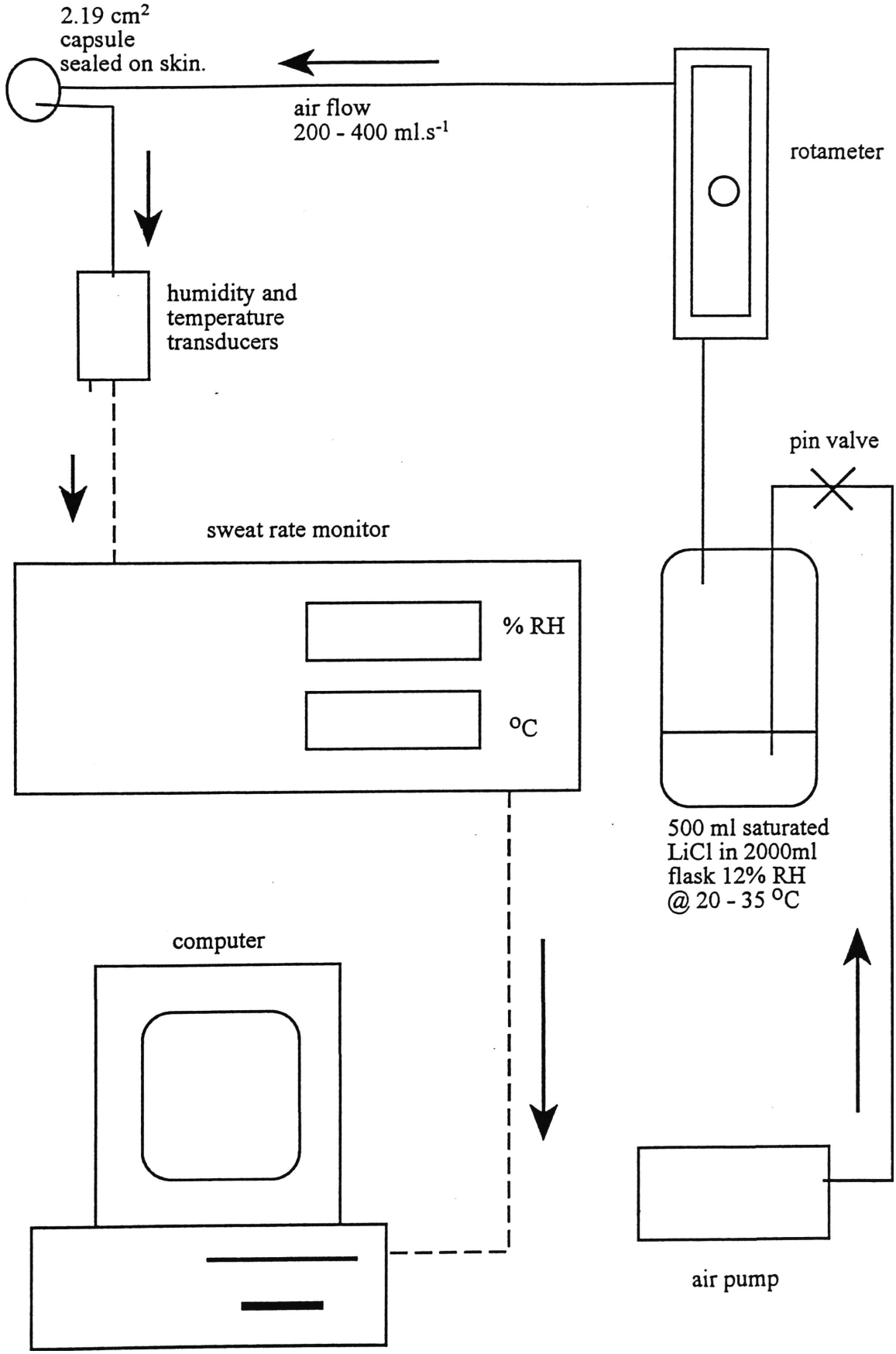


**Figure 3.3:** Schematic of the sweat monitoring system

*3.1.3.3.1 Calibration*

The humidity sensors were calibrated on a weekly basis in 38°C, by introducing a range of standard relative humidities and adjusting the respective channel gains and zero offsets to match these standards. The saturated solutions used for calibration included: lithium chloride (LiCl; 11%), sodium iodide (NaI; 33.5%), sodium chloride (NaCl; 75%), and potassium phosphate ($K_2PO_4$; 93%). Values were recorded across the range of solutions once they stabilised (10 minutes) and the gain and offset adjusted accordingly until the percent relative humidity was displayed accurately across the range. Relevant calibration equations were then constructed by performing analysis on the values attained during calibration against the standard relative humidity values of the saturated solutions. The coefficients of these equations were then used to determine the actual relative humidity values.

Prior to each testing day, a calibration was performed in 38°C using the saturated solutions LiCl, NaI, and NaCl to check for drift in the system. The LiCl standard was attached to the system one hour prior to each experiment and was used as the baseline standard relative humidity for the duration of each test. The increase in relative humidity above 12%, once the sweat capsules were tightly sealed to the skin of a subject, indicated sweat onset. The temperature display was calibrated in different ambient conditions on a monthly basis. A calibrated thermometer (Dobbie Instruments, Dobros total immersion, Australia) was placed near the transducers and the zero offset adjusted once the thermometer reading and the temperature display stabilised, so that they equalled one another.

**3.1.3.3.2 Calculation of sweat rate**

The following equation was used for the calculation of sweat rate:

$$\dot{m}_s = (((RH*PH_2O_a*\dot{V})/(100*T_{cap}*3.464))-((RH_{exp}*PH_2O_a*\dot{V})/(100*T_a*3.464)))/A$$

Where:

$\dot{m}_s$ = mass flow of water off the skin in $g.cm^2.min^{-1}$

RH = relative humidity obtained from the capacitance hygrometer once air has left the skin (%)

$RH_{exp}$ = relative humidity of water vapour entering the capsule at the target experimental temperature (%)

$\dot{V}$ = airflow through the capsule measured from the rotameter ($L.min^{-1}$)

$PH_2O_a$ = partial pressure of water vapour that would be obtained for air entering the capsule if it were 100% saturated (mmHg)

$T_{cap}$ = temperature of air leaving the sweat capsule (°K)

$T_a$ = ambient temperature (°K)

3.464 = (water vapour gas constant)

A = 2.19 $cm^2$

*3.1.3.3.3 Determination of the sweat threshold and sensitivity*

The sweating threshold was determined for each subject at the forehead and forearm, and defined as the continuous sweating threshold. The steps for determining the threshold were as follows:

(i) one second data was graphed against time,

(ii) the median of minutes 1 to 2 was recorded as a representation of baseline (or zero) $\dot{m}_{sw}$,

(iii) a point was chosen where $\dot{m}_{sw}$ began to elevate above the median value for at least 5 minutes without returning to baseline,

(iv) a linear function was fit to 1 minute of data on the baseline prior to this point, and another linear function to the data after it began to elevate,

(v) simultaneous equations were used to obtain the time (seconds) where these functions intercepted, the time was taken as the sweating threshold,

(vi) the $T_{ac}$ that corresponded with this time was taken as $T_{ac}$ threshold for continuous sweating.

In situations where the threshold was difficult to determine, multiple $\dot{m}_{sw}$ sites for the one subject were superimposed for confirmation. If the baseline data was not flat, a larger sample time was taken as a representation of the horizontal linear function

to remove the effects of large deviations. Also, when noise was present at the apparent threshold, the linear function was fit to the rise of the noise, at the first section of the response where elevation began.

Sweat rate sensitivity was determined from 5 minute averaged data of $\dot{m}_{sw}$ against $T_{ac}$. Once obvious plateaux were deleted, a linear function was fit to the remaining data to obtain the slope. When hysteresis was apparent, the 5 minute data was superimposed on 1 second data for visual confirmation of the slope.

### 3.1.3.4 Body Core Temperature

Core temperature was measured at 1 Hz from the auditory canal (aural) during the HST, and recorded manually from the rectum throughout the acclimation phase. $T_{ac}$ was considered as the temperature approximately 1 cm in to the auditory canal, measured using a servo-heated aural thermometer (London Hospital Medical College, Zero-gradient aural thermometer, London). The thermometer monitors the temperature of the aural canal and the outer ear separately, both of which are insulated by a padded headset. A servo-heating circuit warms and maintains the outer ear at the temperature of the aural canal. This system provides a reliable indication of body $T_c$ (Keatinge and Sloan, 1975). $T_{ac}$ data were sampled at 1 Hz, and analog output passed, via an eight channel, 12-bit analog-to-digital converter (Computer Boards Inc., PPIO-A18, Mansfield, U.S.A.), to a mains-isolated IBM compatible laptop computer (Total Peripherals, Notebook 386SX, Sydney, Australia). $T_{re}$ was measured using a thermistor (Yellow Springs Instrument Co., Inc., YSI probe no. 401, Ohio), positioned 12 cm beyond the anal sphincter. Data was recorded manually during acclimation from a tele-thermometer (Yellow Springs Instrument Co., Inc., YSI model 46, Ohio).

These two measurement sites have recently been validated at rest and during cycle ergometry (n=6) across a range of thermal environments (Maw and Taylor, 1992). Core temperatures were monitored during 30 min rest in a thermoneutral environment (24°C), and later during 30 min of continuous lower-body exercise in the same environment, followed by 30 min of exercise in either a cool (13°C) or a hot

(36°C) condition. While $T_{ac}$ was a mean of 0.6°C cooler than oesophageal temperature ($T_{oe}$) during rest, it responded in parallel to $T_{oe}$ throughout exercise, regardless of environmental temperature. $T_{re}$ dissociated from both $T_{oe}$ and $T_{ac}$ during exercise in the cool condition. However, the magnitude of change did not differ significantly and $T_{re}$ rose in parallel to $T_{oe}$ during cycling in the thermoneutral and hot conditions. Figure 3.4A and 3.4B show the responses of the three $T_c$ measurements to thermoneutral exercise and then exercise in either the cool or hot condition.

#### *3.1.3.4.1 Calibration*

All thermistors, including skin, rectal and aural were calibrated in a stirred water bath (Grant Instruments Ltd., Cambridge) against a calibrated referenced mercury thermometer (Dobbie Instruments, Dobros total immersion, Australia) on a monthly basis. All thermistors were grouped together and positioned in open water near the thermometer bulb. The water bath was set at 50°C and thermistor output data collected. Data were logged every 2 minutes in the temperature range of 20 to 42°C for the thermistors and 28 to 40°C for the aural thermometer. The reading time for the Dobros thermometer corresponded with data logging onset every 2 min. Linear analysis was then performed on this data and a calibration equation established for each thermistor. Using the coefficients of the linear equations, thermistor output data was converted to the corrected temperatures.

### 3.1.3.5 Skin Temperature

Skin temperatures were monitored at 0.2 Hz from thermistors (Grant Instruments Ltd., Cambridge) located at eight sites. These sites included: forehead, right scapula, left upper chest, right upper arm, left lower arm, left hand, right anterior thigh, and left calf (ISO, 1992). Measurements were recorded using a data logger (Grant Instruments Ltd., 1200 Series Squirrel, Cambridge) and later downloaded to a computer for storage.

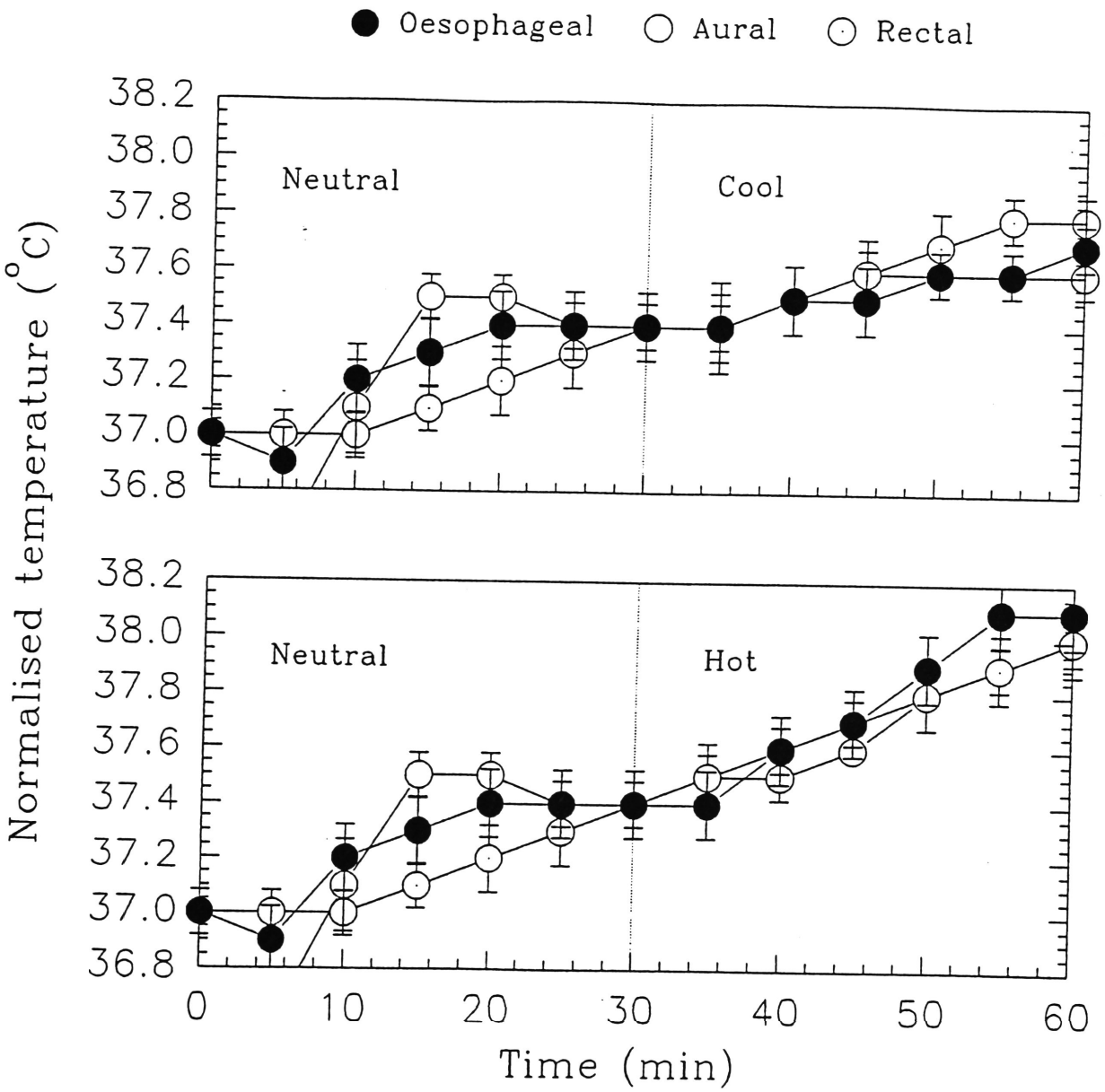


**Figure 3.4:** Oesophageal, aural, and rectal temperatures during 30 minutes of cycle exercise in a thermoneutral environment (24°C), followed by 30 minutes of cycle exercise in either a cool (13°C; A) or a hot (36°C; B) environment (means ± SEM; normalised to mean resting level; from Maw and Taylor, 1992).

These measurements were used in the following equation to determine mean skin temperature ($\overline{T}_{sk}$):$\overline{T}_{sk}$ = (((forehead+right upper arm+left lower arm)*0.07) + ((right scapula+left upper chest)*0.175) + (left hand*0.05) + (right anterior thigh*0.19) + (left calf*0.2)) (ISO, 1992).

### 3.1.3.6 Cardiac frequency

Cardiac frequency was monitored continuously from ventricular depolarisation at 0.2 Hz (Polar Electro SportTester, model PE3000, Finland) and subsequently downloaded to computer for storage. $f_c$, determined using this system, has been validated in our laboratory against a five-lead electrocardiogram (Quinton Q5000; Osborne, 1992). Two subjects remained seated on a bicycle for a period of 10 minutes, then experienced a step increase in work rate to a workload of 150 watts for 17 minutes, followed by a further period of seated rest. A linear regression analysis was performed and it was shown that for submaximal exercise, the SportTester provided a valid indicator of $f_c$ (Figure 3.5).

### 3.1.3.7 Mass and Height

The subject's mass was determined using high resolution platform scales (A&D, Model No. fw-150k, California) calibrated against known mass standards, and height was measured using a stadiometer (Holtain Ltd., Britain). Skinfold measurements were taken with Harpenden callipers.

### 3.1.3.8 Psychophysical variables

The psychophysical variables of thermal sensation and rating of perceived exertion (RPE) were quantified throughout the HST. The thermal sensation scale was a modified version of that produced by Gagge *et al.* (1967), where the end points were extended to enable better resolution of thermal sensation. A recording was taken prior to exposure, upon entering the chamber, and every 5 minutes thereafter. The question was asked "How does the temperature of your body feel?" and subjects answered on

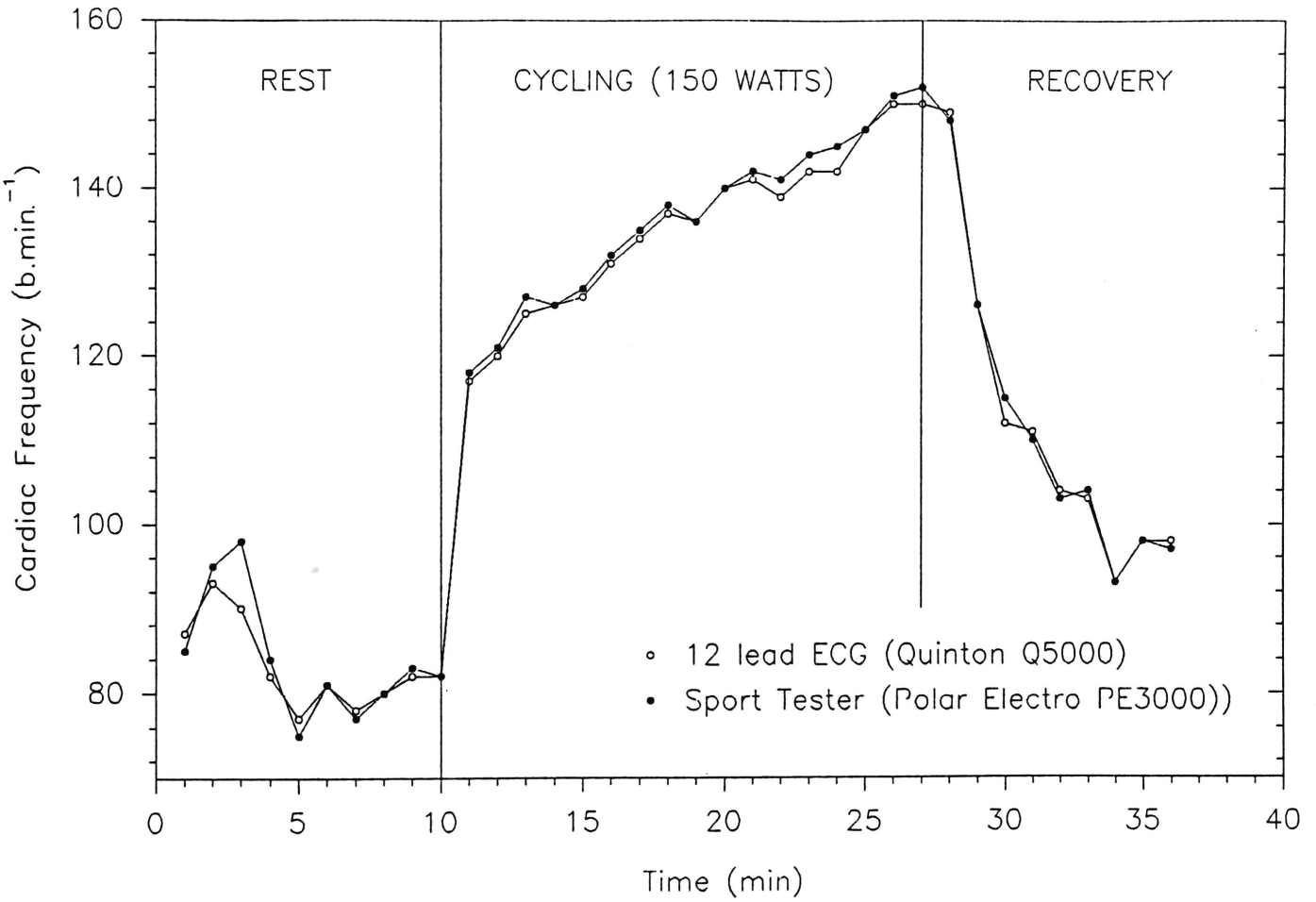


**Figure 3.5:** A comparison of the cardiac frequency determined from the SportTester PE3000 with that obtained simultaneously using a 5-lead ECG during a submaximal exercise step forcing function.

a scale of 1-13. (Appendix D).

Ratings of perceived exertion, for the whole body, chest, and legs began immediately following the commencement of exercise, at 20 minutes, and the question was asked "How hard do you feel you are exercising your whole body/chest/legs".

Subjects gave an answer for each on a scale of 6-20 (Borg, 1962; Appendix E) Differential RPE scores were used since subjects perceive exertion according to the manner in which they experience the stress, as demonstrated by Pandolf (1978). Thus the legs may be perceived as the site for greatest physiological strain in a subject not accustomed to leg exercise.

### 3.1.3.9 Design and Analysis

This study is based on a two by three factorial design. Subjects were nested for factor one (acclimation regime) which had two levels: exercise acclimation; and combined exercise and heat acclimation. Subjects were fully crossed for factor two (heat stress exercise intensity) which had three levels: rest, exercise at 30% $\dot{W}_{peak}$; and exercise at 45% $\dot{W}_{peak}$. Data were averaged over 5 minute intervals ($\pm$ 20 seconds), and analysed using *MANOVA* (Tukey's HSD *post hoc*). Independent t-tests were conducted on data collapsed across time within each of the three phases of the HST. *Alpha* set at the 0.05 level for all analyses.

# CHAPTER FOUR: RESULTS

## 4.1 RESULTS

### 4.1.1 Acclimation

Both neutral physical training, and combined heat and physical training produced greater $\dot{V}_{O2peak}$ values after 10 days of 5.62 $ml.kg^{-1}.min^{-1}$ and 4.38 $ml.kg^{-1}.min^{-1}$ respectively ($P>0.05$). The average work performed each day for both groups is shown in Table 4.1. Work performed during the acclimation regimes, to elevate and maintain $T_{re}$, was significantly higher for the NEUT group ($P<0.05$).

The average time to reach the 1°C elevation in $T_{re}$ was achieved by 29.5 ± 2.7[2] minutes in the NEUT group and 29.2 ± 2.0 minutes in the HEAT group ($P>0.05$). The typical $T_{re}$ response curves for subjects from each group are illustrated in Figure 4.1. Once elevated, the average $T_{re}$ was 37.9°C (± 0.03) and 38.1°C (± 0.05) for the groups respectively (Table 4.2). The change in $T_{re}$ from resting to the elevated temperature was actually significantly greater for the HEAT group ($P<0.05$).

Mean skin temperature averaged 33.2°C (± 0.07) for the NEUT group and 37.4°C (± 0.24) for the HEAT group over the 10 days ($P<0.05$; Table 4.3). Analysis revealed there were no significant differences between the $\overline{T}_{sk}$ recorded for days 1 and 10 of acclimation in either group ($P>0.05$).

Cardiac frequency averaged 133.8 $b.min^{-1}$ (± 1.1) and 143.0 $b.min^{-1}$ (± 2.4) over the 10 days for the NEUT and HEAT groups respectively ($P<0.05$; Table 4.3). There was a significant decrease in $f_c$ for both acclimation groups over time, with the $f_c$ reduction in the HEAT group being greater ($P<0.05$).

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[2] All data are means ± the standard errors of the means, unless otherwise indicated.

**Table 4.1:** Work performed (kilojoules), during two 10-day acclimation regimes undertaken as physical training 22.4°C (RH=41.0%; NEUT), or combined physical training and heat exposure at 38.2°C (RH=39.7%; HEAT).

| DAY | NEUT (n=7) | HEAT (n=7) |
|---|---|---|
| 1 | 485.9 (± 40.2) | 408.9 (± 17.6) |
| 2 | 498.7 (± 39.3) | 399.7 (± 17.3) |
| 3 | 516.4 (± 37.1) | 388.4 (± 25.3) |
| 4 | 507.0 (± 40.8) | 393.4 (± 34.4) |
| 5 | 502.9 (± 61.9) | 393.4 (± 16.0) |
| 6 | 509.5 (± 45.0) | 389.4 (± 36.4) |
| 7 | 503.8 (± 45.2) | 393.7 (± 40.0) |
| 8 | 505.9 (± 43.8) | 404.4 (± 28.5) |
| 9 | 507.3 (± 42.5) | 398.8 (± 40.9) |
| 10 | 511.1 (± 41.3) | 398.8 (± 37.2) |
| mean | 504.9 (± 8.2)* | 396.9 (± 6.5) |

Data are means with standard deviations in parenthesis.

* = significant difference between conditions.

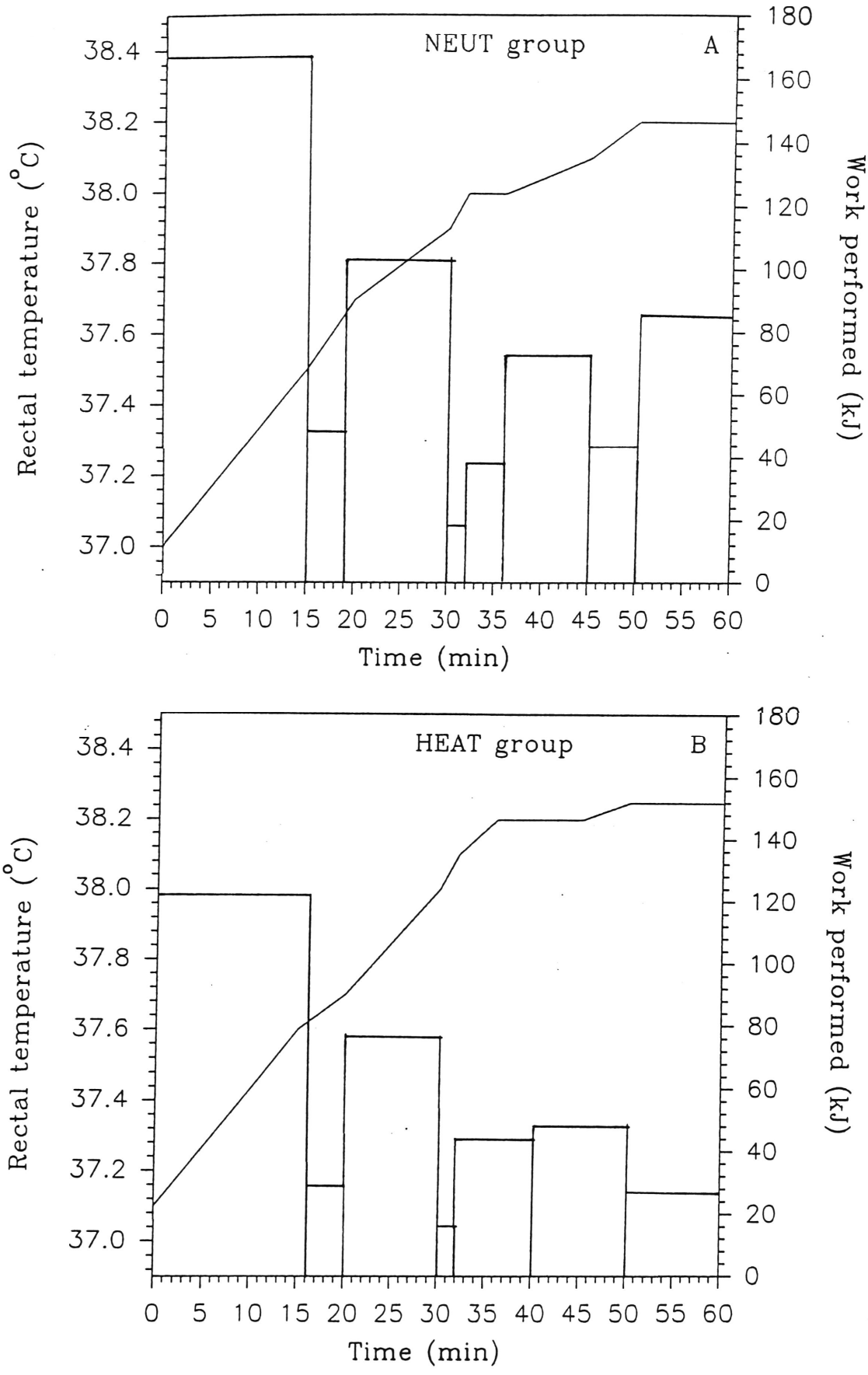


**Figure 4.1:** Typical rectal temperature response curves resulting from work performed (kilojoules) during 1 day of acclimation regimes undertaken by training at 22.4°C (RH=41.0%; NEUT; 4.1A, subject 1), or training at 38.2°C (RH=39.7%; HEAT; 4.1B, subject 1).

**Table 4.2:** Average plateau rectal temperatures, once elevated via endogenous heat production, elicited during a 10-day acclimation regime for subjects physical training at 22.4°C (RH=41.0%; NEUT), or physical training at 38.2°C (RH=39.7%; HEAT). Data also show the change in $T_{re}$ between the pre-exercise resting and plateau values.

| | Elevated $T_{re}$ | | Change in $T_{re}$ | |
|---|---|---|---|---|
| Day | NEUT (n=7) | HEAT (n=7) | NEUT (n=7) | HEAT (n=7) |
| 1 | 37.89 (0.06) | 38.28 (0.13) | 1.07 (0.05) | 0.58 (0.22) |
| 2 | 37.98 (0.06) | 38.24 (0.09) | 1.02 (0.06) | 1.36 (0.10) |
| 3 | 37.91 (0.07) | 38.10 (0.09) | 1.00 (0.05) | 1.37 (0.07) |
| 4 | 37.92 (0.07) | 38.15 (0.08) | 1.03 (0.05) | 1.31 (0.05) |
| 5 | 37.78 (0.07) | 38.03 (0.07) | 1.01 (0.04) | 1.36 (0.11) |
| 6 | 37.78 (0.07) | 37.95 (0.10) | 1.06 (0.04) | 1.34 (0.08) |
| 7 | 37.84 (0.07) | 38.04 (0.12) | 1.08 (0.03) | 1.21 (0.06) |
| 8 | 37.95 (0.06) | 38.0 (0.07) | 1.02 (0.12) | 1.17 (0.06) |
| 9 | 38.01 (0.06) | 37.91 (0.11) | 0.88 (0.15) | 1.14 (0.06) |
| 10 | 37.90 (0.04) | 38.07 (0.09) | 1.08 (0.07) | 1.02 (0.08) |
| mean | 37.90 (0.03)* | 38.08 (0.05) | 1.03 (0.03)* | 1.28 (0.05) |

Data are means with standard errors of the means in parenthesis; * = significant difference between conditions

Abbreviations: $T_{re}$= rectal temperature

**Table 4.3:** Mean skin temperature (°C) and cardiac frequency ($b.min^{-1}$) elicited during a 10-day acclimation regime for subjects training at 22.4°C (RH=41.0%; NEUT), or training at 38.2°C (RH=39.7%; HEAT).

| | Mean skin temperature | | Cardiac frequency | |
|---|---|---|---|---|
| Day | NEUT (n=7) | HEAT (n=7) | NEUT (n=7) | HEAT (n=7) |
| 1 | 33.25 (0.26) | 37.54 (0.28) | 137.35 (2.65) | 154.19 (3.19) |
| 2 | 33.12 (0.24) | 37.94 (0.28) | 134.91 (2.88) | 151.31 (3.61) |
| 3 | 33.51 (0.28) | 37.76 (0.32) | 134.17 (2.9) | 144.16 (2.71) |
| 4 | 33.37 (0.27) | 37.89 (0.27) | 132.29 (3.27) | 145.59 (2.99) |
| 5 | 33.28 (0.3) | 35.74 (1.4) | 127.85 (3.46) | 143.84 (2.65) |
| 6 | 33.04 (0.32) | 37.20 (0.38) | 133.72 (3.25) | 141.33 (2.62) |
| 7 | 33.02 (0.35) | 37.58 (0.31) | 133.01 (3.19) | 140.30 (3.28) |
| 8 | 33.49 (0.3) | 37.69 (0.26) | 137.87 (3.4) | 139.07 (2.81) |
| 9 | 33.18 (0.3) | 37.51 (0.25) | 134.41 (2.64) | 136.19 (2.72) |
| 10 | 33.12 (0.32) | 37.58 (0.59) | 132.27 (3.13) | 134.08 (3.75) |
| mean | 33.24 (0.07)* | 37.44 (0.24) | 133.8 (1.1)* | 143.0 (2.4) |

Data are means with standard errors of the means in parenthesis

* = significant difference between conditions

## 4.2. Heat stress tests

### 4.2.1. Heat stress test one

The typical physiological responses of subjects during the first HST, prior to acclimation, are represented by one subject's data (Figure 4.2 & 4.3). $T_{ac}$ produced varying responses throughout the three phases of the HST. Upon entering the chamber, $T_{ac}$ decreased rapidly by approximately 0.5°C (Figure 4.2A), as a result of moving into the much warmer environment. At approximately 10 minutes, the decrease in $T_{ac}$ slowed, and a period of equilibrium was reached as the body adjusted to the hot environment. At the commencement of the first exercise period (30% $\dot{W}_{peak}$) at 20 minutes, $T_{ac}$ began increasing steadily, resulting from the combined metabolic work and hot environment. Towards the end of this phase, $T_{ac}$ began to plateau again. A slight drop in $T_{ac}$ at the end of this exercise phase was due to exercise being stopped for a short duration to take a SkBF measurement on the stationary thigh. $T_{ac}$ then continued to rise more rapidly during the last exercise phase (45% $\dot{W}_{peak}$), with no indication of a plateau forming. The average increase in $T_{ac}$ for the 60 minute exposure was approximately 1.5°C, with typical body core temperatures of 38.6°C at the conclusion of the test.

In contrast to $T_{ac}$, the commencement of the HST caused $\overline{T}_{sk}$ to begin a progressive rise (Figure 4.2B), which continued until approximately 15 minutes into the resting phase where a plateau was reached. This increase was in the magnitude of 2°C. During the first exercise phase, $\overline{T}_{sk}$ continued to increase slowly before a plateau at about 30 minutes into the HST at approximately 37.3°C. This plateau was maintained briefly into the last exercise phase, where it then increased gradually to 37.9°C.

Thermal sensation showed a similar response to $\overline{T}_{sk}$, since they are related (Figure 4.2C). Thermal sensation increased during the resting phase but reached a plateau at nine (warm) during this phase. The first exercise phase showed further increases in thermal sensation, which continued during phase 3, until a plateau 5

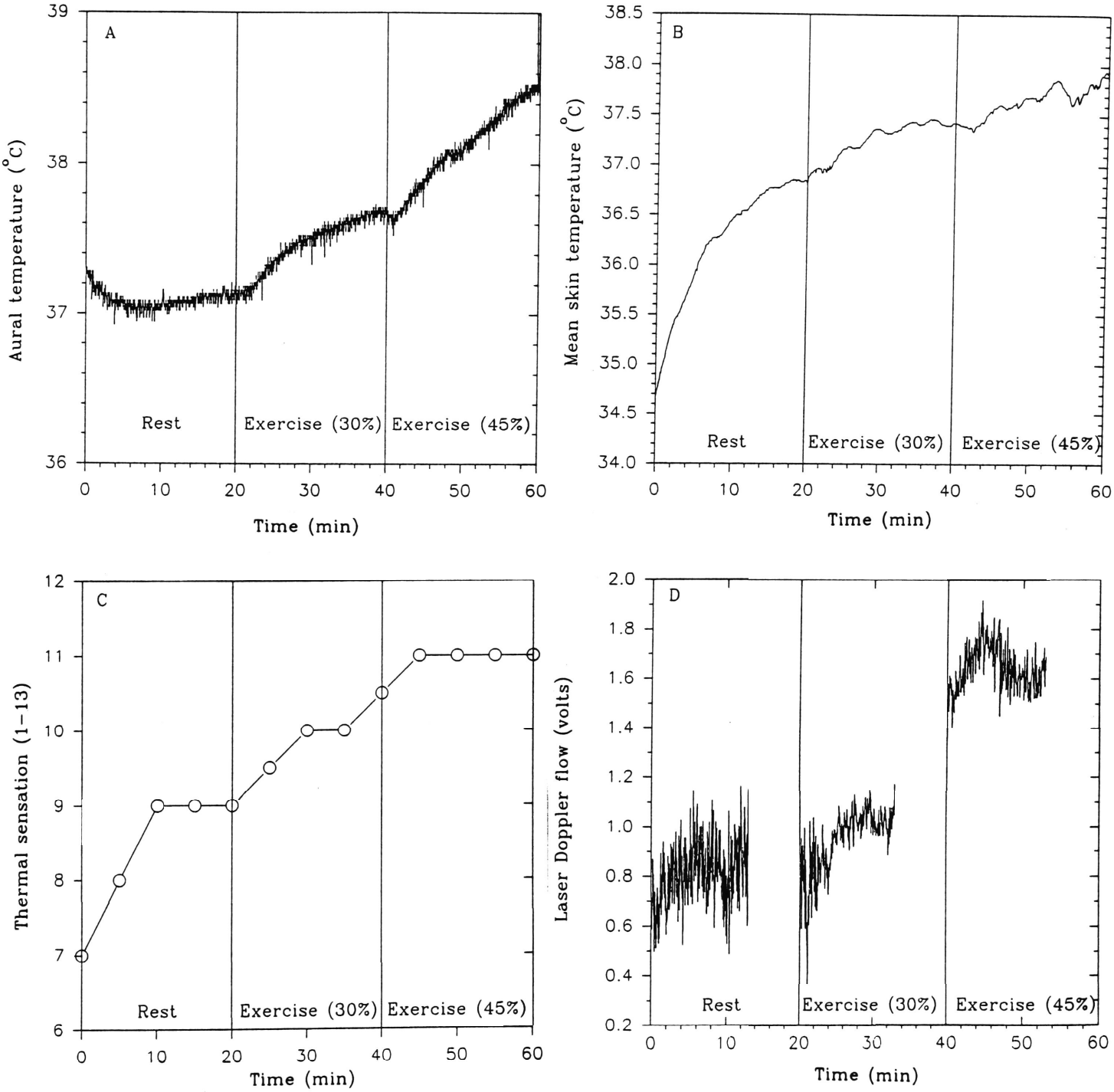


**Figure 4.2:** Typical response data for auditory canal temperature (Figure 4.2A), mean skin temperature (Figure 4.2B), thermal sensation (Figure 4.2C), and forearm skin blood flow (Figure 4.2D) during a 60-min heat stress test (39.8°C, RH 38.6%) before a 10-day thermal acclimation regime (subject 1, NEUT group).

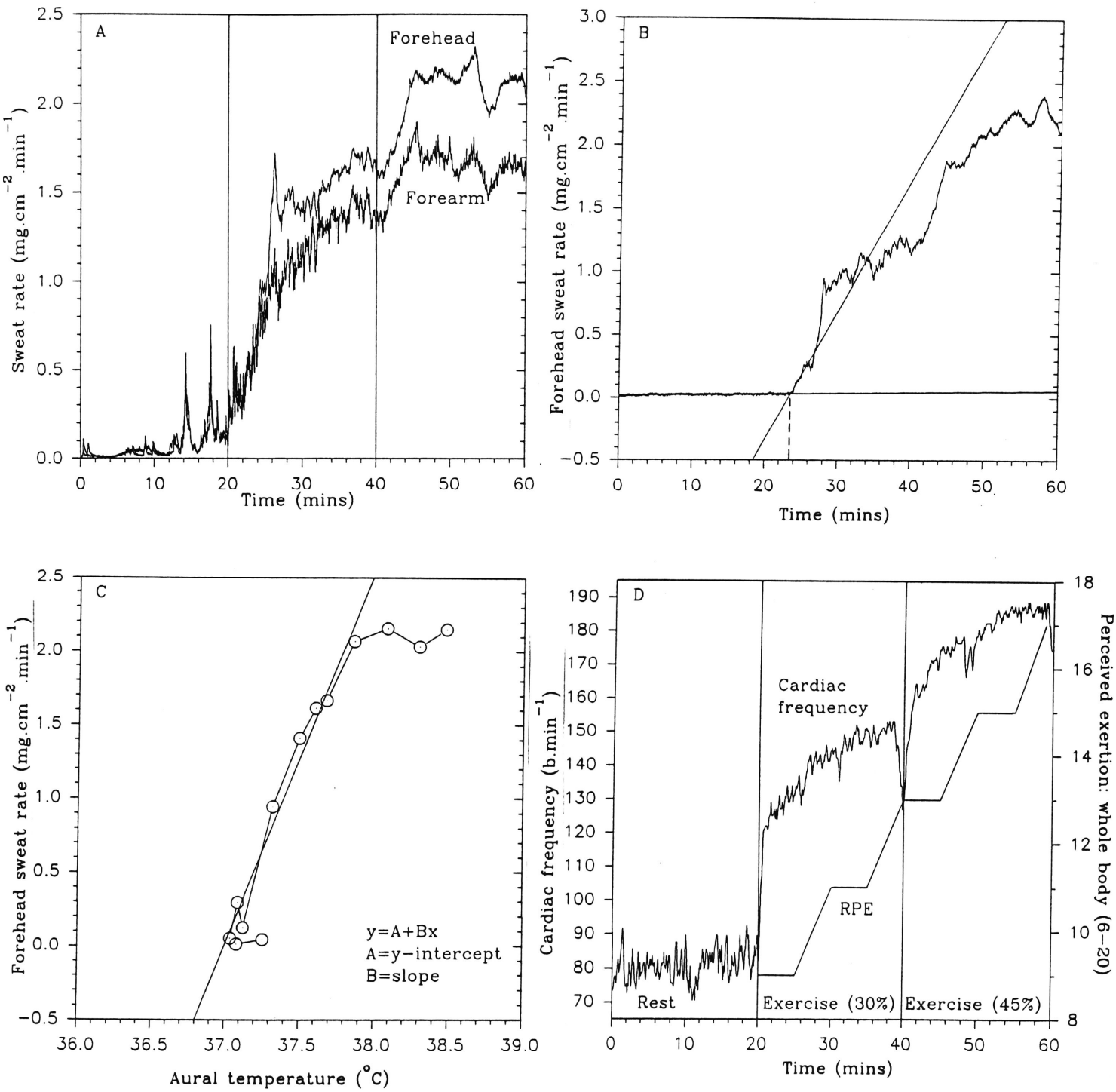


**Figure 4.3:** Typical response data for forehead and forearm sweat rate (Figure 4.3A), sweating threshold (Figure 4.3B) and sensitivity (Figure 4.3C) determination, and cardiac frequency and perceived exertion (Figure 4.3D) during the first 60-min heat stress test (39.8°C, RH 38.6%) before a 10-day thermal acclimation regime (subject 1, NEUT group).

minutes into the last exercise phase at 11 (very hot). This was maintained until the end of the HST.

The LDV data depicted in Figure 4.2D is for the forearm, and was smoothed using a 5 second running average. The last five minutes of each phase was allocated to SkBF measurements at each of the other sites, and the first and last minute of each 15 minute continuous phase of data collection was deleted, so that each phase of the HST consists of 13 minutes of continuous blood flow data at the forearm. LDV showed marked oscillation throughout the HST, particularly during the resting phase. During the resting phase LDV oscillated around an elevating curve, which plateaued, then continued 5 minutes into the first exercise phase. The decrease in SkBF during this phase, could typify exercise onset vasoconstriction. SkBF increased slightly during this phase and reached a plateau of approximately 1.0 volts at 27 minutes. At the commencement of the last exercise phase, SkBF increased very rapidly at first, and then more slowly until about 45 minutes. At this point, SkBF began to decrease before reaching a new plateau level for the remainder of the HST.

Sweat rate, as represented by forehead and forearm $\dot{m}_{sw}$ showed similar response patterns throughout the HST, although the magnitude for sweating became greater at the forehead at approximately 25 minutes into the test (Figure 4.3A). At rest, during the first 10 minutes of the HST, $\dot{m}_{sw}$ oscillated within a narrow range. Towards the latter stages of the resting phase, these oscillations were synchronous and became larger for both the forehead and the forearm, depicting sweat onset during this phase. At the commencement of the first exercise phase, $\dot{m}_{sw}$ increased rapidly at both sites. After approximately 5 minutes into this phase, forehead $\dot{m}_{sw}$ disassociated from forearm $\dot{m}_{sw}$ and continued to increase to a higher level. However, although $\dot{m}_{sw}$ was higher at the forehead than the forearm, changes occurring at the forearm were parallel to the changes in forehead $\dot{m}_{sw}$. The beginning of the last exercise phase showed a further rapid increase in $\dot{m}_{sw}$ at both sites, with plateaux reached at 45 minutes. During the final 10 minutes of the test, $\dot{m}_{sw}$ declined at both sites to a new steady state level by the end of the HST. Typical average $\dot{m}_{sw}$ for the duration of the HST ranged from 1.1 to 2.1 $mg.cm^{-2}.min^{-1}$ for the forehead and 0.7 to 1.3 $mg.cm^{-2}.min^{-1}$ for the forearm.

These values did not relate well to mass change, where values ranged from 6.0 to 9.5 $mg.cm^{-2}.min^{-1}$ for the 60 minute exposure.

The one second data was used to determine the sweating threshold value for all subjects. Figure 4.3B illustrates the method use to determine the $\dot{m}_{sw}$ thresholds. In this example, the threshold point was taken as the point where sweating was elevated above the resting baseline for more than 5 minutes. Linear functions, fit to the baseline and the slope of the rise, determined the time that sweating began. The corresponding $T_{ac}$ was then taken as the threshold value for sweating. Pre-acclimation thresholds ranged from 36.5°C to 37.9°C across both subject groups. The sensitivity of the sweating response was determined from the 5-minute averaged data, illustrated in Figure 4.3C. For determination, a linear function was fit to data following the removal of obvious plateaux. Slope values ranged from 1.4 to 3.9 $mg.cm^{-2}.min^{-1}.°C$ prior to acclimation across both groups.

Cardiac frequency oscillated throughout the three phases of the HST (Figure 4.3D). During the resting phase, $f_c$ maintained a plateau. At the commencement of the first exercise phase, $f_c$ showed a sharp elevation, which was followed by a slower rise while oscillating, approaching a plateau towards the end of this phase. The rapid decrease in $f_c$ was due to a transient pause in exercise so that the LDV measurement could be taken on the stationary thigh. Another sharp rise accompanied the commencement of the final exercise phase, which gradually plateaued at approximately 185 $b.min^{-1}$ with 10 minutes remaining. In this subject, the terminal $f_c$ represented 91% of maximum $f_c$. While RPE is typically linked to $f_c$ by a factor of ten, the scores reported for RPE remained lower than the recorded $f_c$, possibly due to the increased thermal drive accompanying exercise in hot environments.

Although subjects were matched for aerobic power and the sum of 6 skinfolds, analyses of the pre-acclimation HST data revealed differences between the apparent acclimation states of the two subject groups (Table 4.4). $T_{ac}$ and $\overline{T}_{sk}$ were significantly lower for the NEUT group across all phases of the first HST ($P < 0.05$). $f_c$ was

**Table 4.4:** Comparison of auditory canal temperature, mean skin temperature, cardiac frequency, and sweat rate for the forehead and forearm between NEUT and HEAT acclimation groups, during a 60-min heat stress test (39°C, RH 38.6%) before two separate 10-day acclimation regimes.

| HST phase<br>NEUT | $T_{ac}$ (°C) | $\overline{T}_{sk}$ (°C) | $f_c$<br>(b.min$^{-1}$) | $\dot{m}_{sw}$<br>(forehead) | $\dot{m}_{sw}$<br>(forearm) |
|---|---|---|---|---|---|
| 1 | 37.23* | 36.13* | 80.81* | 0.33* | 0.24 |
| 2 | 37.20* | 37.24* | 136.91 | 1.86* | 1.27* |
| 3 | 37.95 | 37.72* | 171.37 | 2.79* | 1.71* |
| mean | 37.44* | 36.96* | 125.94 | 1.56* | 1.01* |
| SEM | 0.07 | 0.10 | 4.49 | 0.12 | 0.07 |
| HEAT | | | | | |
| 1 | 37.62 | 36.67 | 86.75 | 0.15 | 0.19 |
| 2 | 37.52 | 37.83 | 136.29 | 1.34 | 0.99 |
| 3 | 38.23 | 38.45 | 171.60 | 2.05 | 1.41 |
| mean | 37.78 | 37.57 | 128.10 | 1.10 | 0.81 |
| SEM | 0.06 | 0.12 | 4.27 | 0.12 | 0.06 |

Data are means with standard errors of the means

* = significant difference between acclimation conditions.

Abbreviations: $T_{ac}$= auditory canal temperature; $\overline{T}_{sk}$= mean skin temperature; $f_c$= cardiac frequency; $\dot{m}_{sw}$= sweat rate (mg.min$^{-1}$.cm$^{-2}$)

significantly lower during the resting phase of the HST for the NEUT group ($P < 0.05$). Both forehead and forearm $\dot{m}_{sw}$ were significantly greater for the NEUT group across all phases of the HST. These findings suggest that the NEUT group possessed a higher state of acclimatisation than the HEAT group prior to the acclimation regimes.

### 4.2.2. Pre-versus post-acclimation heat strain

A clear acclimation effect was apparent, with $T_{ac}$ data reflecting significantly lower physiological strain following the HEAT condition (Figure 4.4A & 4.4B). This change was not apparent in the NEUT condition. Analyses of the data, collapsed across the three phases of the HST, revealed that the HEAT condition significantly lowered $T_{ac}$ during each phase and for the total exposure ($P < 0.05$). However, the NEUT condition showed no significant differences during any phase of the HST ($P > 0.05$).

Forearm SkBF showed no heat adaptation response following either the NEUT or HEAT exposure (Figure 4.5A & 4.5B). No significant differences were found during any phase of the HST for either of the groups ($P > 0.05$). Of the five intermittently measured sites (Figure 4.6A & 4.6B), at each of the three phases of the HST, forehead SkBF was greater than at all other sites measured, except the upper back during the 30% exercise phase of the pre-acclimation HST for the NEUT group ($P > 0.05$). During the final 20 minutes of the HST, forehead SkBF was significantly lower following HEAT acclimation ($P < 0.05$). The lower SkBF in the HEAT condition produced a significant difference between the NEUT and HEAT regimes following acclimation, during the final phase of the HST ($P < 0.05$). The same pattern of SkBF distribution was observed between phases of the HST and between acclimation regimes. SkBF was lower in the upper back during the second phase of the HST following NEUT acclimation ($P < 0.05$), however, the change was not significant between conditions ($P > 0.05$).

Mean skin temperature data reflected an acclimation effect, being significantly lower across all phases of the HST following the HEAT condition (Figure 4.7A & 4.7B; $P < 0.05$). The NEUT condition produced significantly higher $\overline{T}_{sk}$ for the total

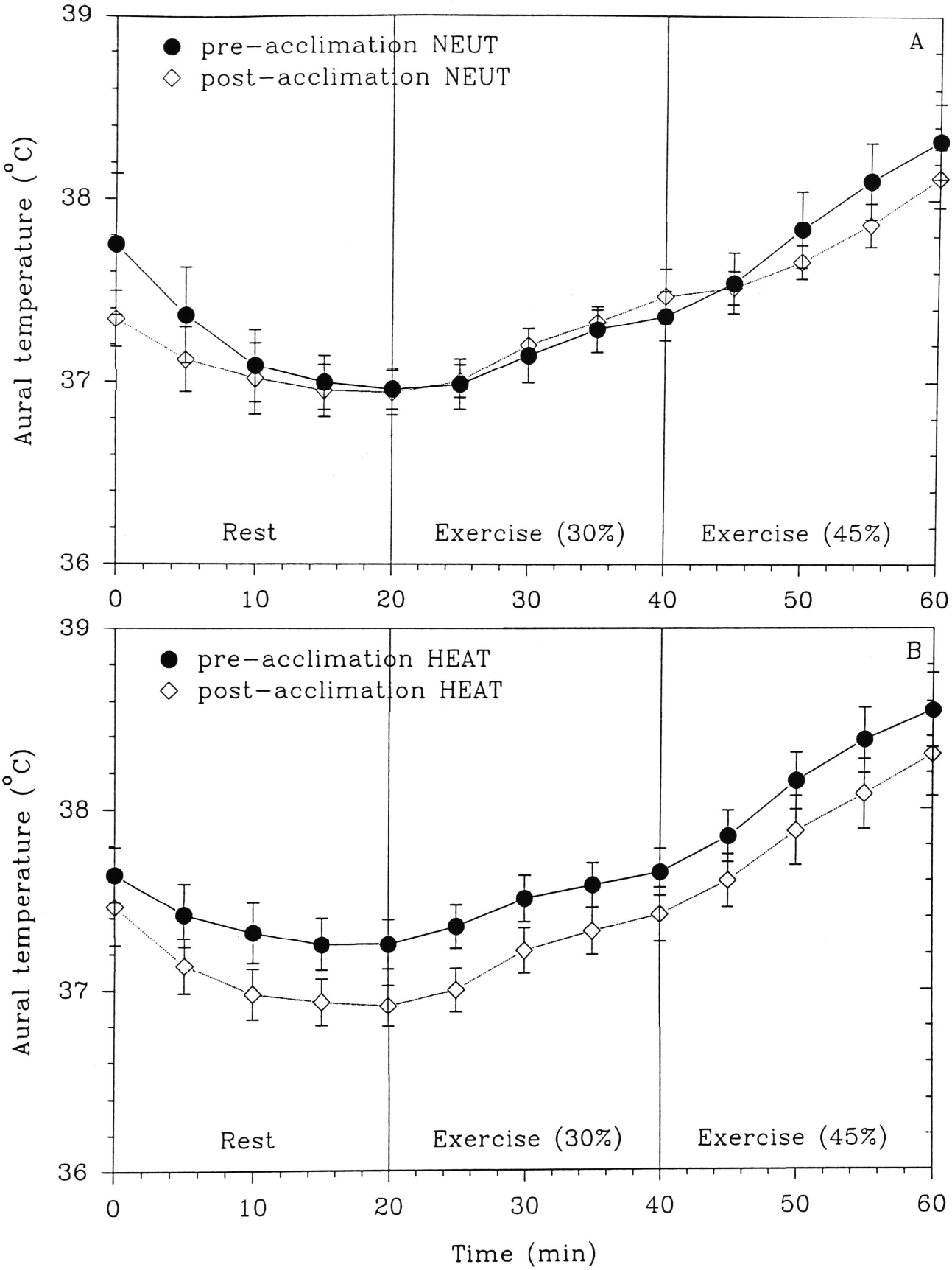


**Figure 4.4:** Auditory canal temperature during a 60-min heat stress test (39.8°C, RH 38.6%) before and after two different 10-day thermal acclimation regimes: physical training at 22.4°C (RH 41.0%; Figure 4.4A); and combined physical training and heat acclimation at 38.2°C (RH 39.7%; Figure 4.4B). Data are means with standard errors of the means.

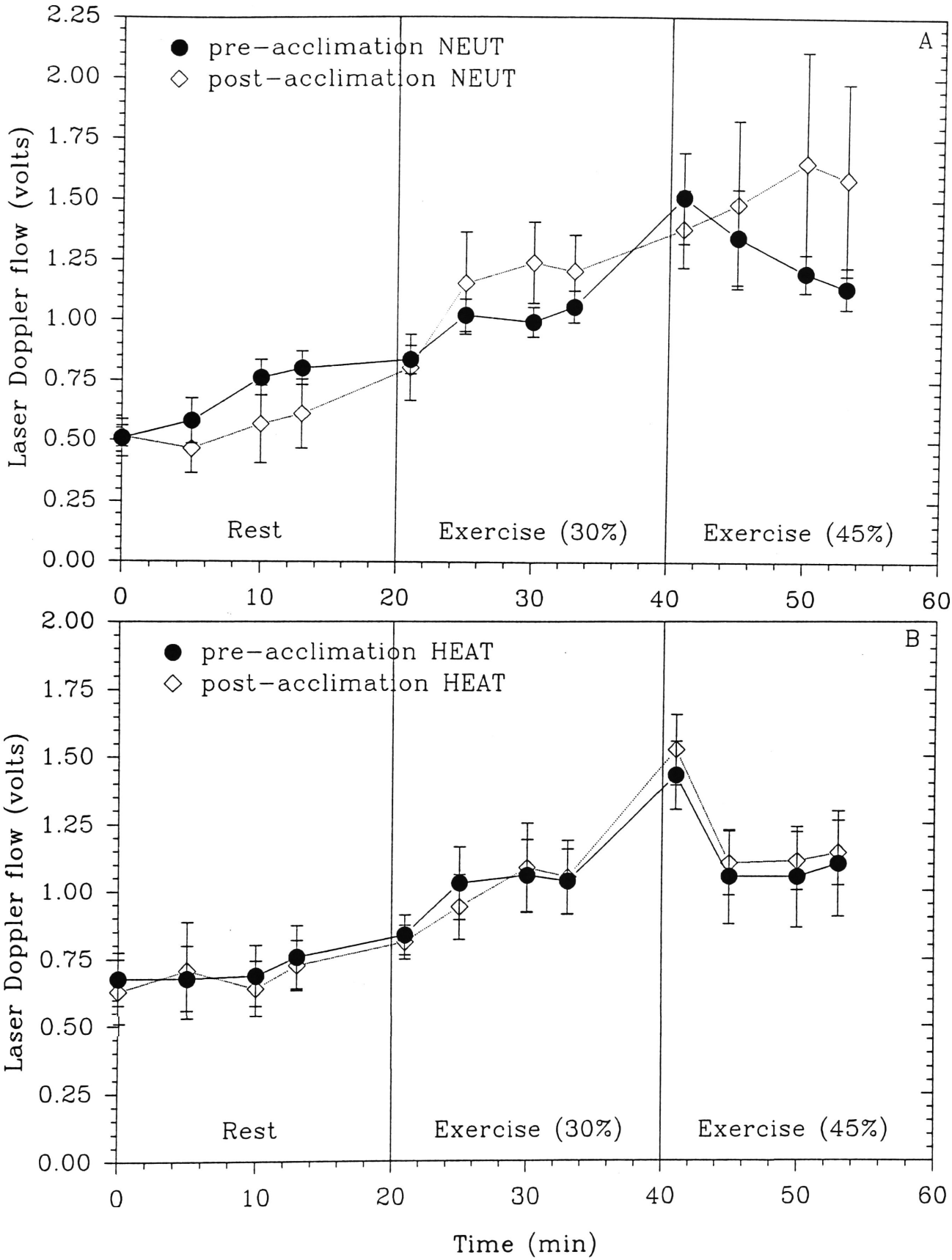


**Figure 4.5:** Forearm skin blood flow during a 60-min heat stress test (39.8°C, RH 38.6%) before and after two different 10-day thermal acclimation regimes: physical training at 22.4°C (RH 41.0%; Figure 4.5A); and combined physical training and heat acclimation at 38.2°C (RH 39.7%; Figure 4.5B). Data are means with standard errors of the means.

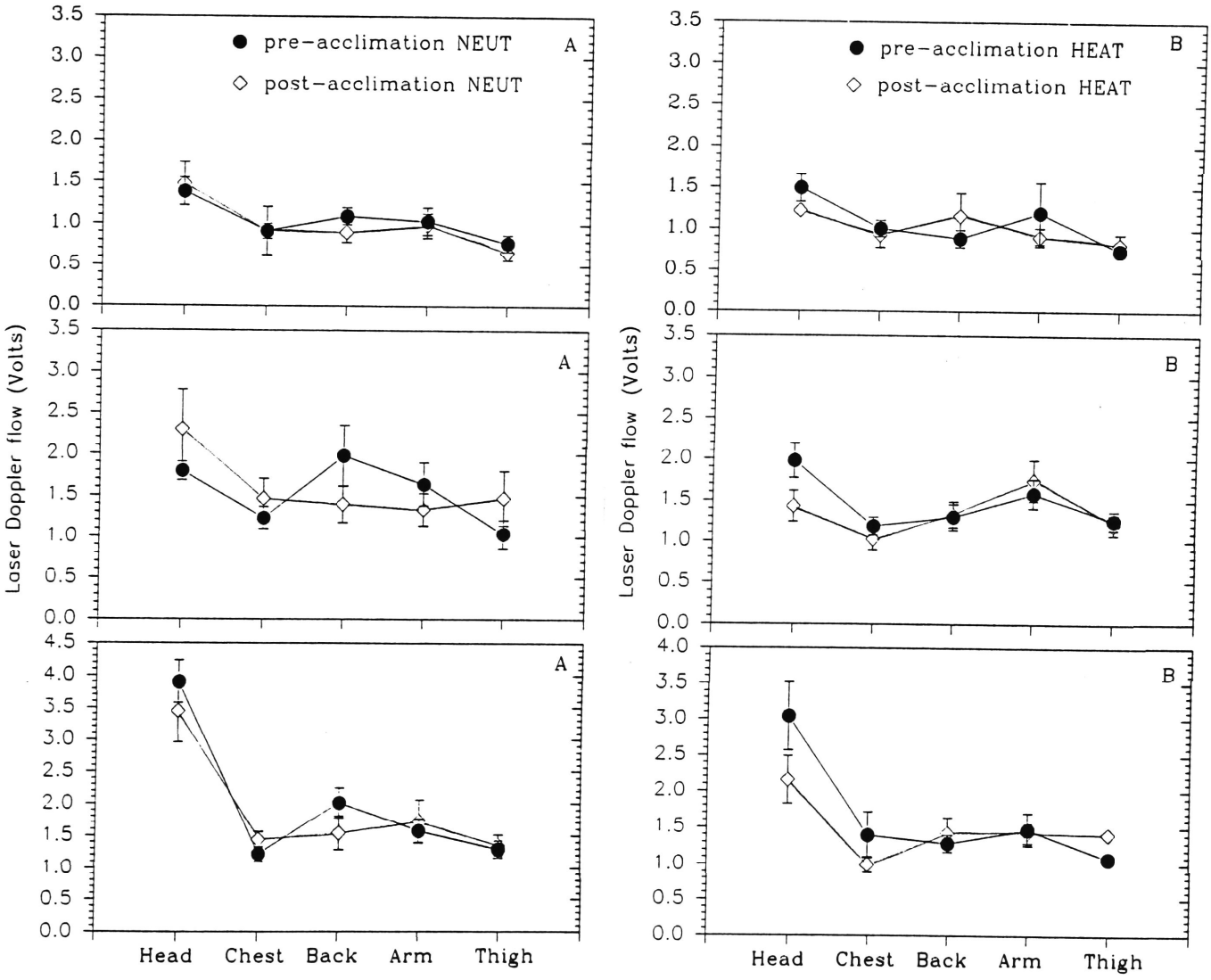


**Figure 4.6:** Skin blood flow at five sites, measured during the final five minutes of each of three 20-minute phases of a 60-min heat stress test (39.8°C, RH 38.6%) before and after two different 10-day thermal acclimation regimes: physical training at 22.4°C (RH 41.0%; Figure A); and combined physical training and heat acclimation at 38.2°C (RH 39.7%; Figure B). Data are means with standard errors of the means.

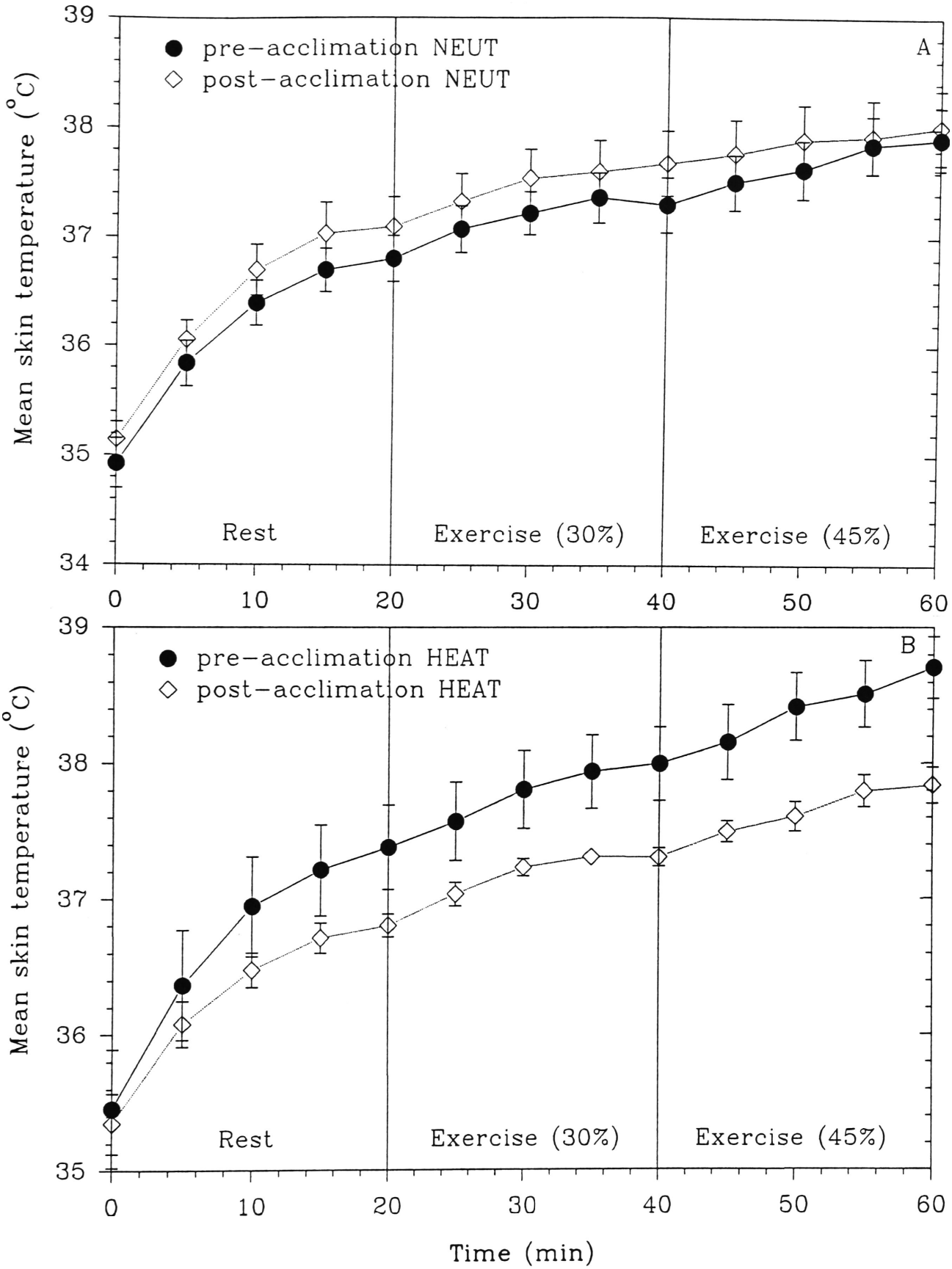


**Figure 4.7:** Mean skin temperature during a 60-min heat stress test (39.8°C, RH 38.6%) before and after two different 10-day thermal acclimation regimes: physical training at 22.4°C (RH 41.0%; Figure 4.7A); and combined physical training and heat acclimation at 38.2°C (RH 39.7%; Figure 4.7B). Data are means with standard errors of the means.

exposure and across the first and second HST phases ($P<0.05$), but not across the final phase. The mean $T_c:\overline{T}_{sk}$ gradient increased from 0.1°C to 0.4°C in the HEAT group and decreased from 0.4°C to 0.1°C in the NEUT group following the acclimation regimes. Thermal sensation was significantly lower across all phases of the HST following both the NEUT and HEAT acclimation ($P<0.05$; Figure 4.8A & 4.8B).

A clear acclimation effect was apparent for $f_c$ following both the NEUT and HEAT acclimation regimes (Figure 4.9A & 4.9B). Analyses revealed that the NEUT and HEAT conditions significantly lowered $f_c$ across all phases of the HST ($P<0.05$). RPE for the whole body was significantly lower across all phases of the HST ($P<0.05$; Figure 4.10A & 4.10B) following HEAT acclimation. The NEUT condition produced no significant differences for the whole-body rating during any phase of the HST ($P>0.05$). RPE for the chest was found to be significantly lower for the HEAT condition, and significantly higher for the NEUT condition during the final phase of the HST ($P<0.05$; Figure 4.11A & 4.11B). RPE scores from the legs revealed significantly lower effort sense during both exercise phases for the HEAT condition ($P<0.05$; Figure 4.12B). No apparent difference was found following NEUT acclimation ($P>0.05$; Figure 4.12A).

Both the HEAT and NEUT conditions produced acclimation effects on $\dot{m}_{sw}$. Analyses indicated that during the first 40 minutes of the second HST, forehead $\dot{m}_{sw}$ was equivalent for the heat acclimated subjects ($P>0.05$), but lower for the neutral group ($P<0.05$; Figure 4.13A & 4.13B). This indicates a more efficient sweating response for the NEUT group, since $T_{ac}$ remained essentially the same (Figure 4.4A). However, the HEAT group, with lower post-acclimation $T_{ac}$ produced more prolific sweating. During the resting phase of the HST, there were no significant differences in forearm $\dot{m}_{sw}$ ($P>0.05$; Figure 4.14A & 4.14B) for the NEUT or HEAT conditions. However, during the final 20 minutes of the HST, the heat acclimated group produced a significantly greater $\dot{m}_{sw}$ relative to control ($P<0.05$). Forearm $\dot{m}_{sw}$ was significantly higher in the second and final phases for the HEAT condition ($P<0.05$), but a significantly higher forearm $\dot{m}_{sw}$ was only apparent for the final phase of the HST for the NEUT condition ($P<0.05$).

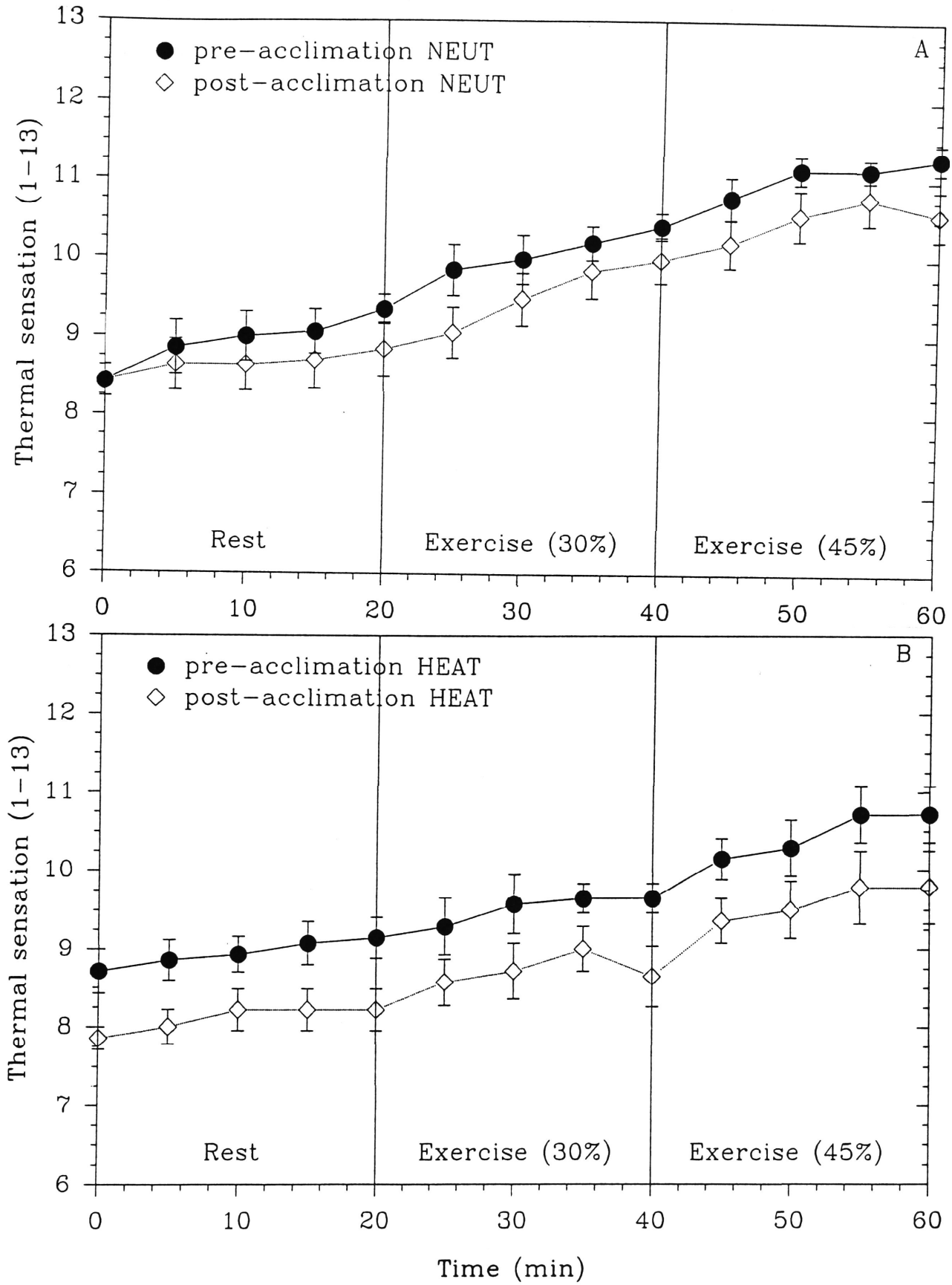


**Figure 4.8:** Thermal sensation during a 60-min heat stress test (39.8°C, RH 38.6%) before and after two different 10-day thermal acclimation regimes: physical training at 22.4°C (RH 41.0%; Figure 4.8A); and combined physical training and heat acclimation at 38.2°C (RH 39.7%; Figure 4.8B). Data are means with standard errors of the means.

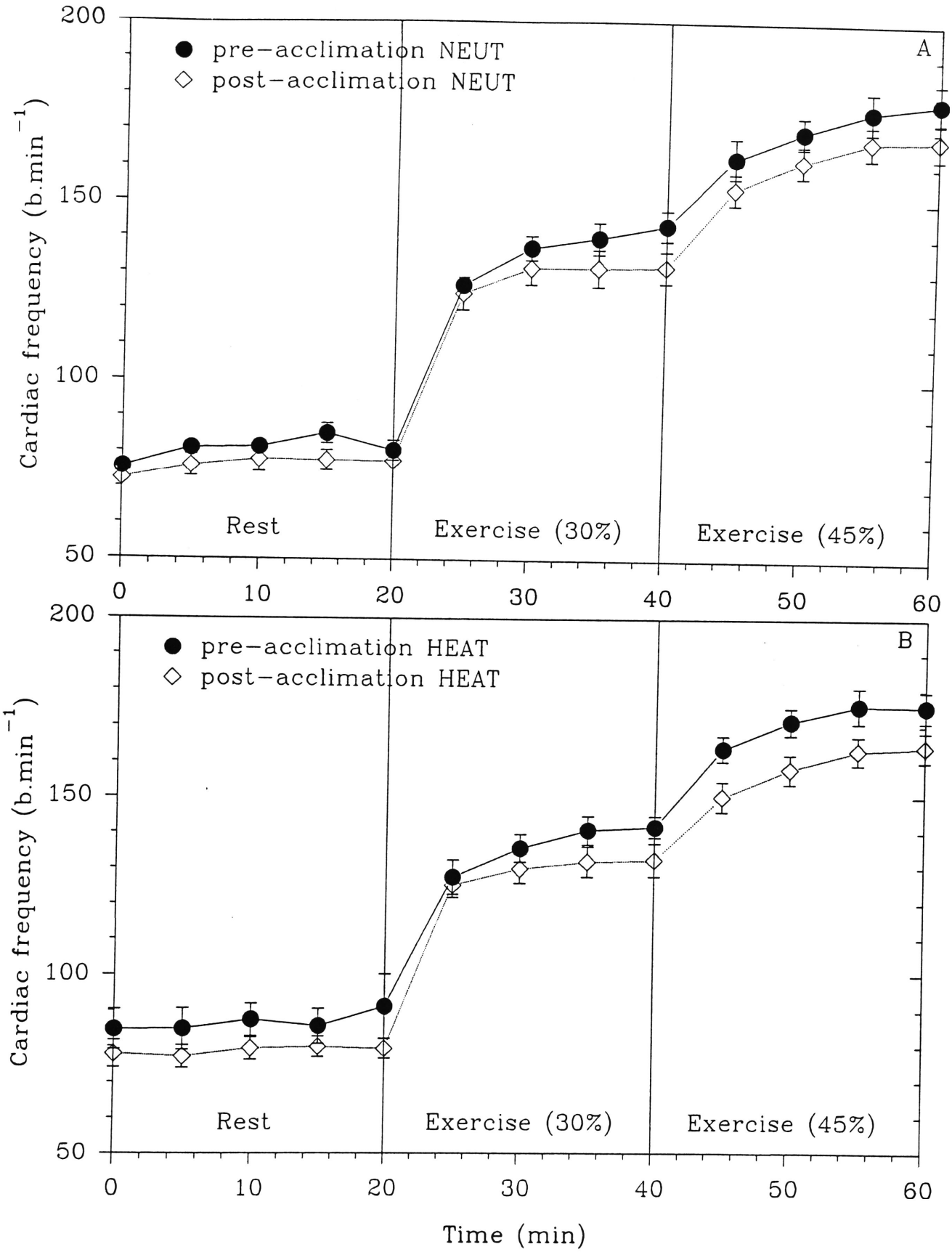


**Figure 4.9:** Cardiac frequency during a 60-min heat stress test (39.8°C, RH 38.6%) before and after two different 10-day thermal acclimation regimes: physical training at 22.4°C (RH 41.0%; Figure 4.9A); and combined physical training and heat acclimation at 38.2°C (RH 39.7%; Figure 4.9B). Data are means with standard errors of the means.

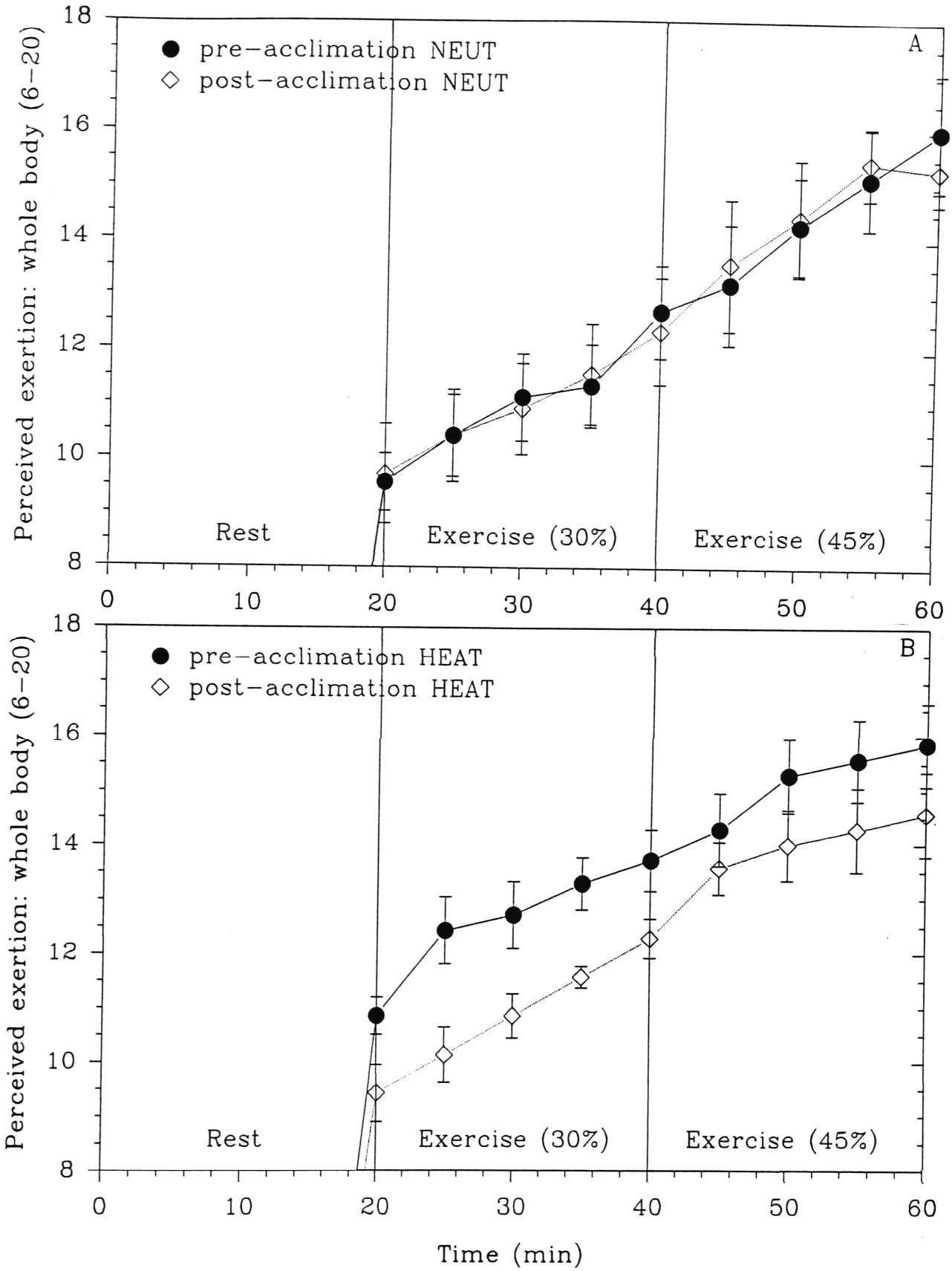


**Figure 4.10:** Rating of perceived exertion (whole body) during a 60-min heat stress test (39.8°C, RH 38.6%) before and after two different 10-day thermal acclimation regimes: physical training at 22.4°C (RH 41.0%; Figure 4.10A); and combined physical training and heat acclimation at 38.2°C (RH 39.7%; Figure 4.10B). Data are means with standard errors of the means.

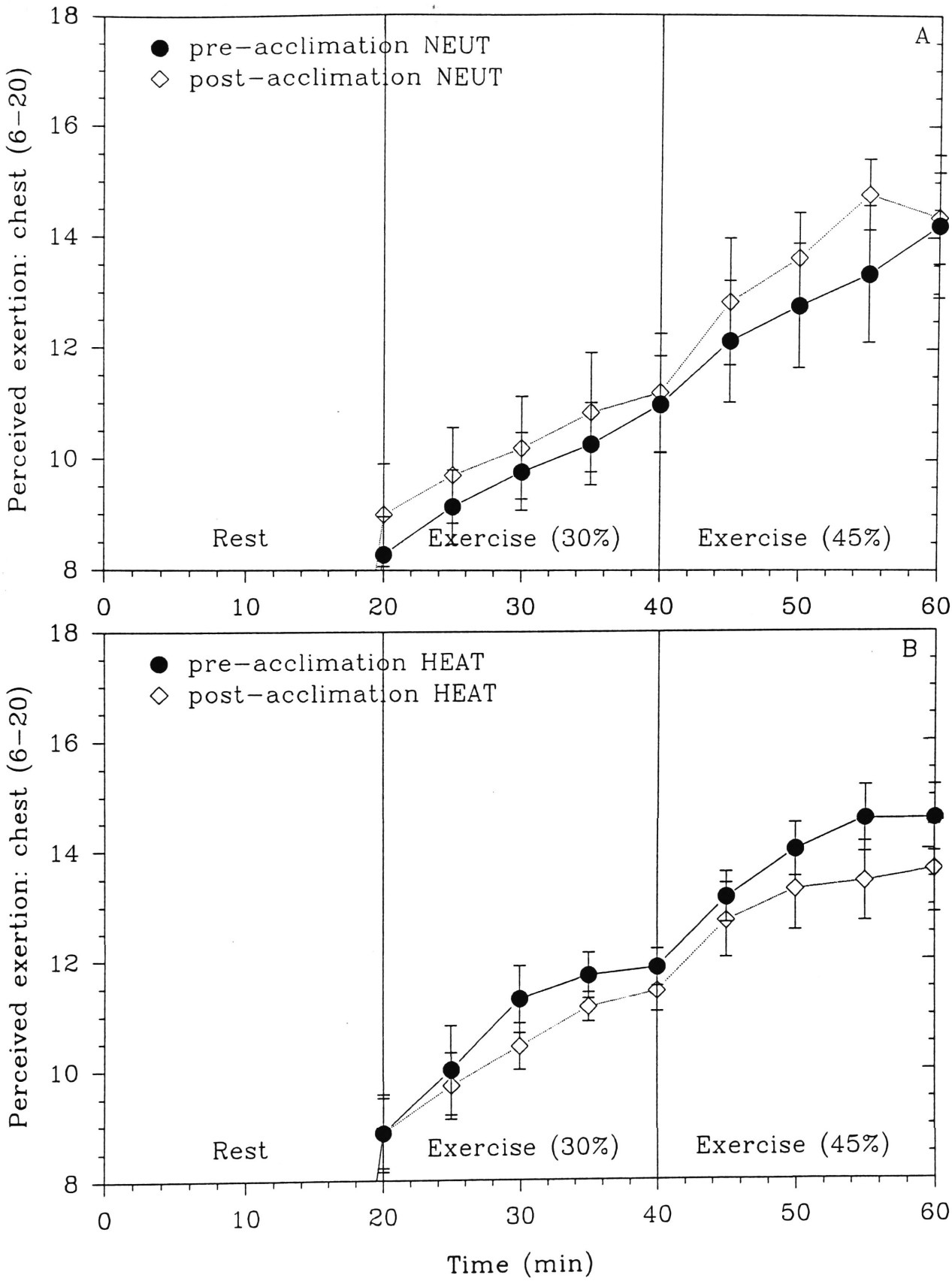


**Figure 4.11:** Rating of perceived exertion (chest) during a 60-min heat stress test (39.8°C, RH 38.6%) before and after two different 10-day thermal acclimation regimes: physical training at 22.4°C (RH 41.0%; Figure 4.11A); and combined physical training and heat acclimation at 38.2°C (RH 39.7%; Figure 4.11B). Data are means with standard errors of the means.

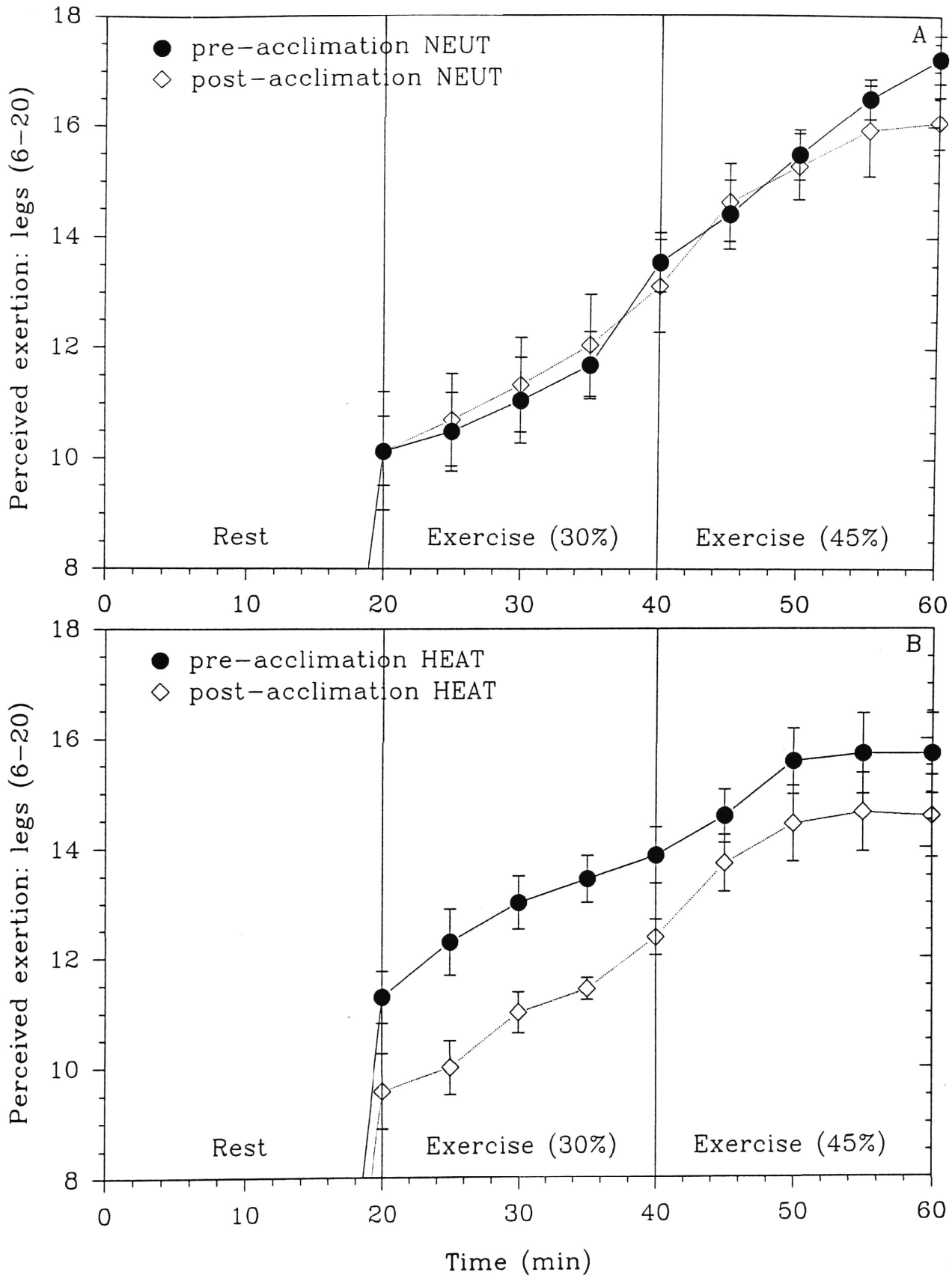


**Figure 4.12:** Rating of perceived exertion (legs) during a 60-min heat stress test (39.8°C, RH 38.6%) before and after two different 10-day thermal acclimation regimes: physical training at 22.4°C (RH 41.0%; Figure 4.12A); and combined physical training and heat acclimation at 38.2°C (RH 39.7%; Figure 4.12B). Data are means with standard errors of the means.

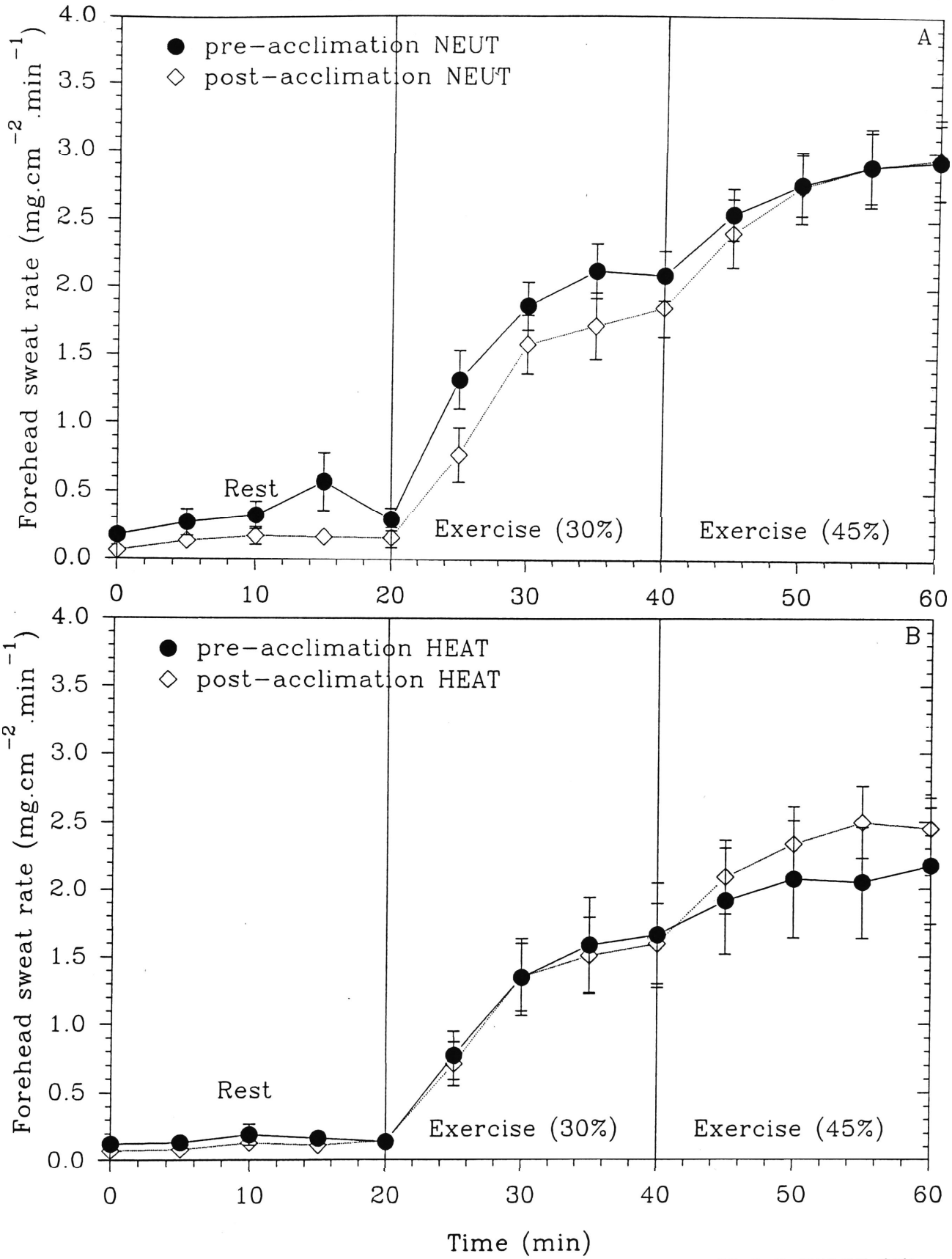


**Figure 4.13:** Forehead sweat rate during a 60-min heat stress test (39.8°C, RH 38.6%) before and after two different 10-day thermal acclimation regimes: physical training at 22.4°C (RH 41.0%; Figure 4.13A); and combined physical training and heat acclimation at 38.2°C (RH 39.7%; Figure 4.13B). Data are means with standard errors of the means.

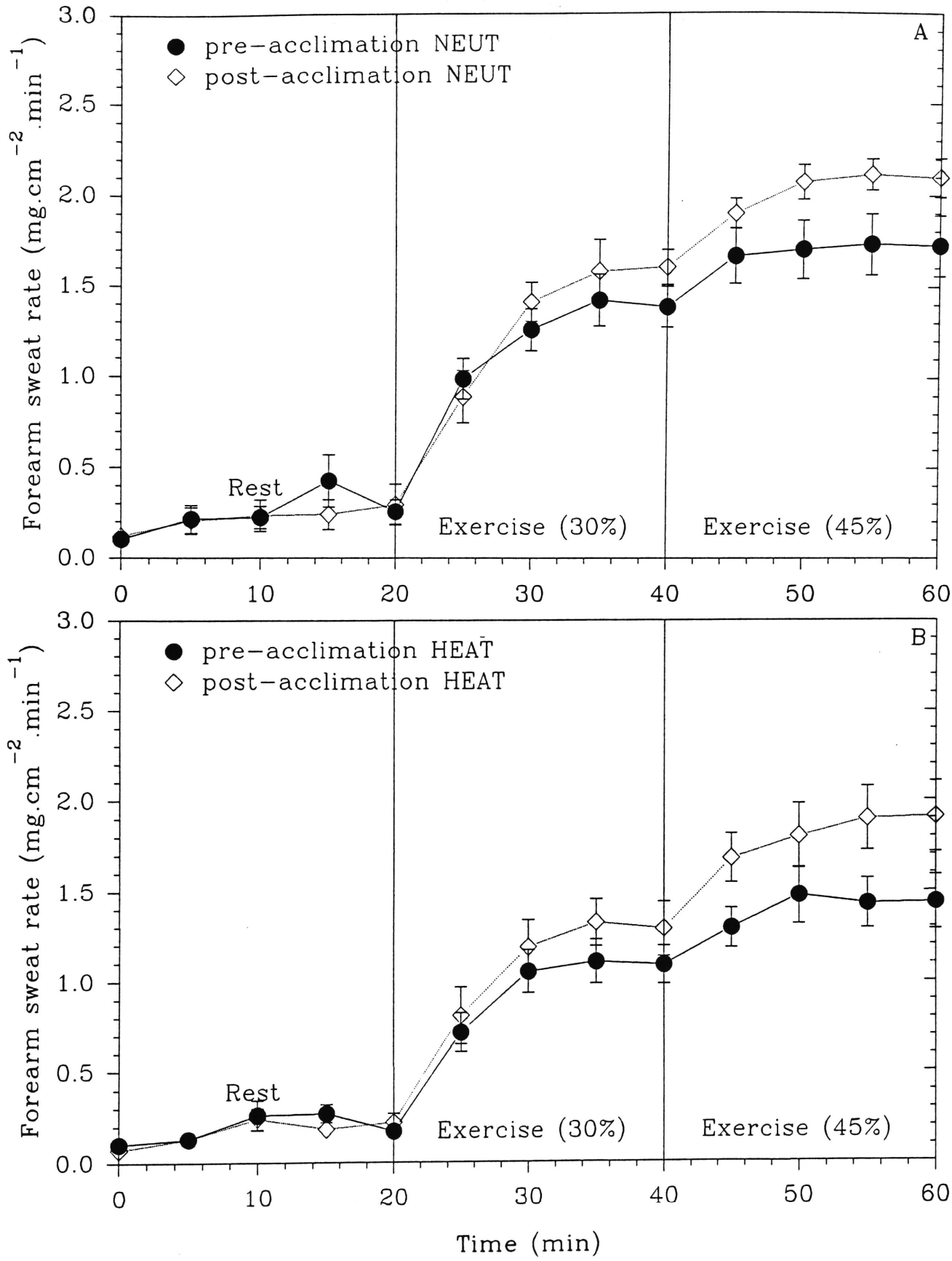


**Figure 4.14:** Forearm sweat rate during a 60-min heat stress test (39.8°C, RH 38.6%) before and after two different 10-day thermal acclimation regimes: physical training at 22.4°C (RH 41.0%; Figure 4.14A); and combined physical training and heat acclimation at 38.2°C (RH 39.7%; Figure 4.14B). Data are means with standard errors of the means.

Both NEUT and HEAT acclimation regimes decreased the $T_{ac}$ threshold for sweating, however, these changes were not significantly different ($P > 0.05$). The average decrease in the sweat threshold, associated with the NEUT condition, was 0.32°C for the forehead and 0.28°C for the forearm (Table 4.5). While the respective decreases associated with the HEAT condition were 0.55°C and 0.62°C. $\overline{T}_{sk}$ at the sweating threshold increased slightly for the forehead and forearm in the NEUT condition, with the average increases being 0.39°C for the forehead and 0.53°C for the forearm ($P > 0.05$; Table 4.6). In contrast, the HEAT condition produced average decreases of 0.29°C and 0.45°C in $\overline{T}_{sk}$ at the sweating threshold ($P > 0.05$; Table 4.6).

Sweat rate sensitivity increased for both the NEUT and HEAT conditions, however, these differences were not significant ($P > 0.05$; Table 4.7). For all subjects, the average increase in sensitivity associated with NEUT acclimation was 0.66 $mg.min^{-1}.cm^{-2}.°C^{-1}$ for the forehead and 0.78 $mg.min^{-1}.cm^{-2}.°C^{-1}$ for the forearm. The increases associated with HEAT acclimation was 0.32 $mg.min^{-1}.cm^{-2}.°C^{-1}$ and 0.13 $mg.min^{-1}.cm^{-2}.°C^{-1}$ respectively.

Subjects lost 0.89 $\pm$ 0.16 and 0.99 $\pm$ 0.16 for the physical training and heat acclimation groups respectively, in the heat stress tests preceding acclimation regimes. Following acclimation subjects lost 0.76 $\pm$ 0.12 and 0.94 $\pm$ 0.32 respectively. These differences were not significant in either group following acclimation ($P > 0.05$; Figure 4.15A & 4.15B).

On the basis of the physiological adaptations following the acclimation regimes, acclimation may have occurred in both groups. Following acclimation in the HEAT condition, subjects showed decreased physiological strain as a result of adaptations in the following variables: decreased $T_{ac}$, $f_c$, $\overline{T}_{sk}$, perceived exertion, and thermal sensation.' Sweating became more prolific, particularly during the final phase of the HST for the forehead, and the second and final phases for the forearm. The NEUT portrayed decreased physiological strain through decreased $f_c$ and thermal sensation. Forehead sweating became more efficient as a result of decreased sweat required to maintain the same $T_{ac}$ in the second HST. Also, forearm $\dot{m}_{sw}$ increased during the

**Table 4.5:** Sweating thresholds (°C) obtained during a 60-min heat stress test (39.8°C, RH 38.6%), administered before and after a 10-day acclimation for subjects training 22.4°C (RH=41.0%; NEUT), or training at 38.2°C (RH=39.7%; HEAT).

| NEUT | Pre heat stress test | | Post heat stress test | |
|---|---|---|---|---|
| Subject | Forehead | Forearm | Forehead | Forearm |
| 1 | 37.14 | 37.12 | 36.72 | 36.65 |
| 2 | 37.14 | 37.14 | 36.62 | 36.55 |
| 3 | 36.90 | 36.88 | 37.49 | 37.49 |
| 4 | 36.72 | 36.65 | 36.84 | 36.81 |
| 5 | 37.95 | 37.93 | 36.98 | 36.96 |
| 6 | 36.45 | 36.45 | 37.11 | 37.23 |
| 7 | 38.80 | 38.92 | 37.08 | 37.47 |
| mean | 37.30 (0.31) | 37.30 (0.32) | 36.98 (0.12) | 37.02 (0.38) |
| HEAT | | | | |
| 1 | 37.24 | 37.24 | 37.12 | 37.19 |
| 2 | 36.88 | 36.88 | 37.48 | 37.69 |
| 3 | 36.88 | 37.09 | 36.75 | 36.78 |
| 4 | 39.23 | 39.23 | 37.03 | 37.37 |
| 5 | 37.35 | 38.02 | 36.77 | 36.81 |
| 6 | 37.32 | 37.44 | 36.60 | 36.55 |
| 7 | 37.42 | 37.54 | 36.69 | 36.67 |
| mean | 37.47 (0.30) | 37.63 (0.30) | 36.92 (0.12) | 37.01 (0.16) |

Standard errors of the means are in parenthesis.

**Table 4.6:** Mean skin temperature (°C) at the sweating thresholds, obtained during a 60-min heat stress test (39.8°C, RH 38.6%), administered before and after a 10-day acclimation for subjects training 22.4°C (RH=41.0%; NEUT), or training at 38.2°C (RH=39.7%; HEAT).

| NEUT | Pre heat stress test | | Post heat stress test | |
|---|---|---|---|---|
| Subject | Forehead | Forearm | Forehead | Forearm |
| 1 | 36.77 | 36.77 | 37.71 | 37.67 |
| 2 | 37.44 | 36.77 | 37.94 | 37.87 |
| 3 | 35.68 | 34.96 | 37.53 | 37.53 |
| 4 | 36.60 | 36.59 | 36.43 | 36.18 |
| 5 | 36.21 | 36.21 | 36.39 | 36.09 |
| 6 | 35.94 | 35.85 | 35.88 | 35.81 |
| 7 | 35.96 | 35.81 | 35.45 | 35.45 |
| mean | 36.37 (0.23) | 36.14 (0.25) | 36.76 (0.37) | 36.67 (0.38) |
| HEAT | | | | |
| 1 | 35.60 | 35.63 | 36.13 | 36.13 |
| 2 | 35.88 | 35.90 | 36.54 | 36.12 |
| 3 | 36.58 | 36.28 | 36.45 | 35.81 |
| 4 | 37.01 | 36.97 | 37.08 | 36.52 |
| 5 | 37.77 | 37.74 | 36.75 | 36.43 |
| 6 | 37.93 | 37.93 | 36.76 | 36.75 |
| 7 | 37.77 | 37.30 | 36.80 | 36.80 |
| mean | 36.93 (0.36) | 36.82 (0.34) | 36.64 (0.11) | 36.37 (0.14) |

Standard errors of the means are in parenthesis.

**Table 4.7:** Sensitivity of the sweating response ($mg.min^{-1}.cm^{-2}.°C^{-1}$) obtained during a 60-min heat stress test (39.8°C, RH 38.6%), administered before and after a 10-day acclimation for subjects training 22.4°C (RH=41.0%; NEUT), or training at 38.2°C (RH=39.7%; HEAT).

| NEUT | Pre heat stress test | | Post heat stress test | |
|---|---|---|---|---|
| Subject | Forehead | Forearm | Forehead | Forearm |
| 1 | 2.54 | 2.10 | 1.61 | 1.54 |
| 2 | 2.55 | 1.58 | 1.76 | 1.31 |
| 3 | 1.37 | 1.13 | 1.42 | 0.85 |
| 4 | 3.44 | 4.41 | 3.66 | 4.24 |
| 5 | 2.65 | 1.64 | 4.10 | 3.84 |
| 6 | 3.94 | 2.03 | 4.42 | 3.98 |
| 7 | 1.17 | 0.58 | 5.34 | 3.20 |
| mean | 2.52 (0.38) | 1.92 (0.46) | 3.19 (0.59) | 2.71 (0.54) |
| HEAT | | | | |
| 1 | 7.20 | 1.84 | 3.69 | 3.91 |
| 2 | 1.79 | 0.98 | 5.28 | 1.92 |
| 3 | 4.11 | 3.48 | 5.56 | 2.09 |
| 4 | 1.33 | 1.13 | 0.92 | 1.19 |
| 5 | 1.82 | 2.29 | 2.97 | 2.65 |
| 6 | 2.91 | 2.85 | 2.72 | 1.65 |
| 7 | 0.67 | 1.08 | 0.87 | 1.17 |
| mean | 2.83 (0.84) | 1.95 (0.37) | 3.14 (0.71) | 2.08 (0.36) |

Standard errors of the means are in parenthesis.

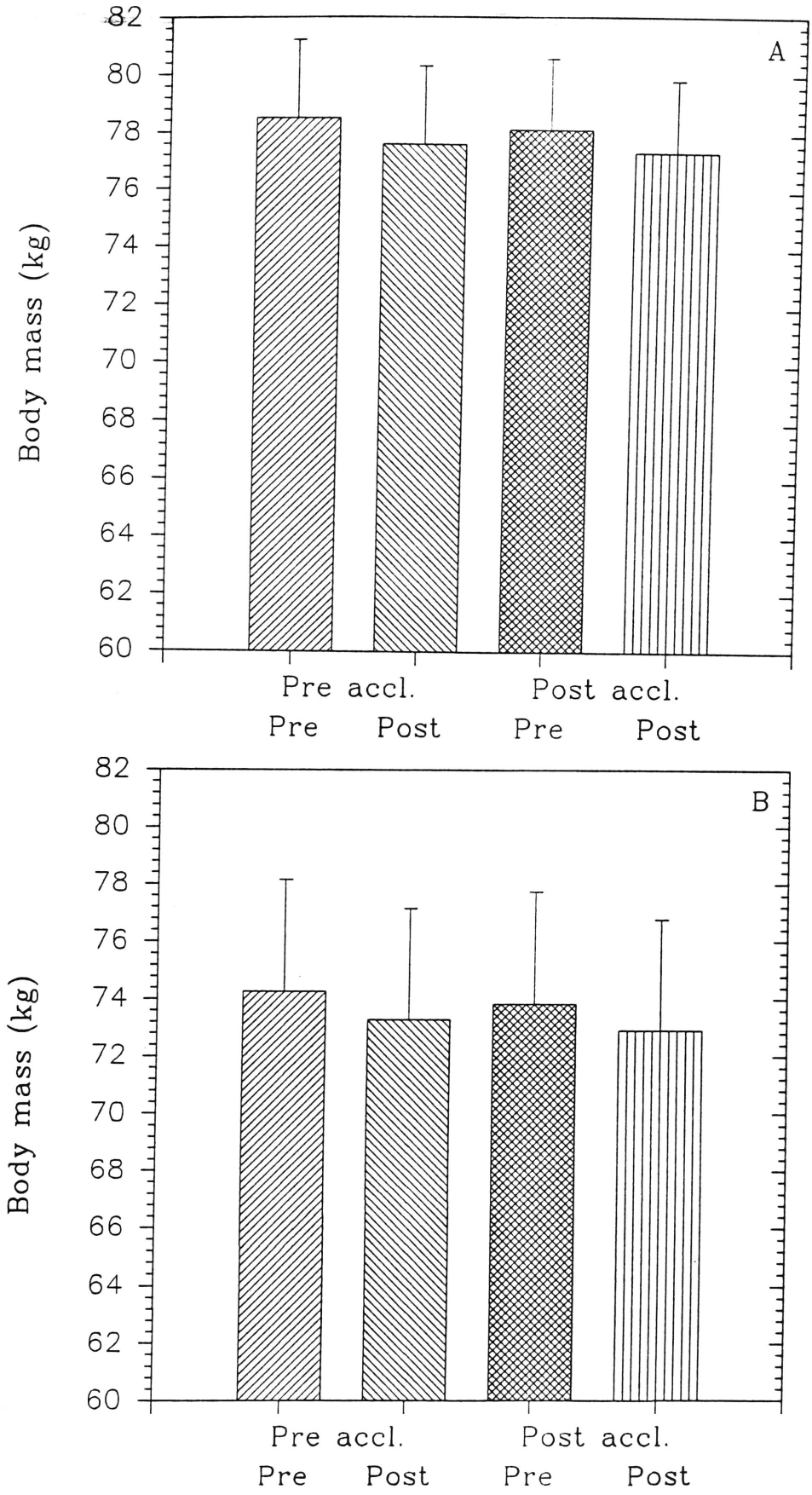


**Figure 4.15:** Body mass (kg) during a 60-min heat stress test (39.8°C, RH 38.6%) before and after separate 10-day thermal acclimation regimes: physical training at 22.4°C (RH 41.0%) and combined physical training and heat acclimation at 38.2°C (RH 39.7%). Data are means with standard errors of the means.

second and final phases of the HST. Decreased physiological strain was more apparent following the HEAT condition, however, this may have been a function of the NEUT group having greater acclimatisation prior to the acclimation regime.

### 4.2.3. Comparisons between acclimation protocols

Physiological strain was reduced in both the NEUT and HEAT acclimation regimes in differing proportions. The effectiveness of each acclimation protocol was compared by analysing the extent of the physiological adaptations for each variable following both regimes. This was achieved by computing an average value for each variable over the four time periods during the HST: rest, 30% $\dot{W}_{peak}$, 45% $\dot{W}_{peak}$, and the total exposure, and then subtracting the pre-HST value from the post-HST value. The resultant value described the acclimation-induced change in a particular variable in each period. Comparisons of the change scores between the two acclimation regimes could then be made (Table 4.8 & 4.9).

Both the NEUT and HEAT acclimation conditions showed decreases in $T_{ac}$ over the total HST time period, however, this decrease was significantly greater following the HEAT condition ($P<0.05$), with the NEUT condition showing only very slight decreases. These between condition differences were significant for all periods except during the 45% $\dot{W}_{peak}$ time period.

Skin blood flow showed virtually no change over the total exposure time period ($P>0.05$). It was slightly reduced during the resting time period for both conditions and during the first 30% $\dot{W}_{peak}$ time period for the HEAT condition ($P>0.05$). The first 30% $\dot{W}_{peak}$ and 45% $\dot{W}_{peak}$ time periods for the NEUT group and the 45% $\dot{W}_{peak}$ time period for the HEAT group produced slight increases in SkBF, although these changes were not significantly different between acclimation regimes ($P>0.05$).

During each of the four time periods of the HST, $\overline{T}_{sk}$ increased following NEUT, and decreased following HEAT acclimation. The magnitude of this change was greater in each period following the HEAT condition ($P<0.05$). Thermal sensation

**Table 4.8:** Physiological adaptations elicited following separate 10-day acclimation regimes for subjects training at 22.4°C (RH=41.0%; NEUT), or training at 38.2°C (RH=39.7%; HEAT). Acclimation is represented by the change scores, computed from the difference between the pre- and post-acclimation heat stress test data.

| Variable | Condition | Rest | 30% $\dot{W}_{peak}$ | 45% $\dot{W}_{peak}$ | Total |
|---|---|---|---|---|---|
| $T_{ac}$ (°C) | NEUT | -0.2 (0.1)* | 0.1 (0.1)* | -0.2 (0.1) | -0.1 (0.1)* |
| | HEAT | -0.5 (0.1) | -0.3 (0.1) | -0.3 (0.1) | -0.4 (0.1) |
| LDV | NEUT | -0.1 (0.1) | 0.1 (0.1) | 0.2 (0.2) | 0.1 (0.1) |
| (volts) | HEAT | -0.02 (0.1) | -0.02 (.04) | 0.1 (0.1) | 0.01 (0.1) |
| $\overline{T}_{sk}$ (°C) | NEUT | 0.3 (0.1)* | 0.3 (0.1)* | 0.2 (0.1)* | 0.3 (0.1)* |
| | HEAT | -0.4 (0.2) | -0.6 (0.1) | -0.8 (0.1) | -0.6 (0.1) |
| Thermal | NEUT | -0.3 (0.1)* | -0.5 (0.1) | -0.6 (0.1) | -0.4 (0.1)* |
| Sensation | HEAT | -0.8 (0.1) | -0.8 (0.1) | -0.9 (0.1) | -0.8 (0.1) |
| $f_c$ | NEUT | -6.1 (1.0) | -7.8 (1.6) | -11.1 (1.2) | -8.2 (0.8) |
| ($b.min^{-1}$) | HEAT | -8.9 (1.8) | -5.8 (1.5) | -11.2 (1.8) | -8.6 (1.0) |
| Perceived | NEUT | --- | 0.04 (0.3)* | -0.1 (0.3)* | -.02 (0.2)* |
| exertion | HEAT | --- | -1.8 (0.3) | -1.2 (0.2) | -1.5 (0.2) |

Data are means with standard errors of the means in parenthesis

* = significant difference between acclimation conditions.

Abbreviations: $T_{ac}$= auditory canal temperature; LDV= laser-Doppler velocimetry; $\overline{T}_{sk}$= mean skin temperature; $f_c$= cardiac frequency; $\dot{W}_{peak}$= peak work rate; ---= data not available.

**Table 4.9:** Physiological adaptations elicited following separate 10-day acclimation regimes for subjects training at 22.4°C (RH=41.0%; NEUT), or training at 38.2°C (RH=39.7%; HEAT). Acclimation is represented by the change scores, computed from the difference between the pre- and post-acclimation heat stress test data.

| Variable | Condition | Rest | 30% $\dot{W}_{peak}$ | 45% $\dot{W}_{peak}$ | Total |
|---|---|---|---|---|---|
| $\dot{m}_{sw}$ forehead | NEUT | -0.2 (0.1)* | -0.4 (0.1)* | -.03 (0.1)* | -0.2 (.04)* |
| | HEAT | -0.04 (.02) | -0.1 (0.1) | 0.3 (0.1) | 0.1 (0.04) |
| Threshold (°C) | NEUT | --- | --- | --- | -0.3 (0.3) |
| | HEAT | --- | --- | --- | -0.6 (0.4) |
| Sensitivity | NEUT | --- | --- | --- | 0.7 (0.7) |
| | HEAT | --- | --- | --- | 0.3 (0.8) |
| $\dot{m}_{sw}$ forearm | NEUT | -.03 (.04)* | 0.1 (0.1) | 0.3 (0.1) | 0.1 (0.04) |
| | HEAT | -.02 (.02) | 0.2 (0.1) | 0.4 (0.1) | 0.2 (0.04) |
| Threshold (°C) | NEUT | --- | --- | --- | -0.3 (0.3) |
| | HEAT | --- | --- | --- | -0.6 (0.4) |
| Sensitivity | NEUT | --- | --- | --- | 0.8 (0.5) |
| | HEAT | --- | --- | --- | 0.1 (0.5) |
| Mass (kg) | NEUT | --- | --- | --- | -0.1 (0.1) |
| | HEAT | --- | --- | --- | -0.1 (0.1) |

Data are means with standard error of the means in parenthesis; * = significant difference between conditions. Abbreviations: $\dot{m}_{sw}$= sweat rate in $ml.cm^{-2}.min^{-1}$; Slope= $ml.cm^{-2}.min^{-1}.°C^{-1}$; kg= kilograms; $\dot{W}_{peak}$= peak work rate; ---= data not available.

decreased in the total time period for both groups, however, the decrease was significantly greater following the HEAT condition ($P<0.05$). The other three time periods of the HST also showed decreases in thermal sensation, but the change was only significantly greater during the resting time period ($P<0.05$).

Cardiac frequency decreased in all four time periods for both the NEUT and HEAT conditions. However, these changes were not significant between groups for any of these periods ($P>0.05$). RPE showed decreases across all time periods for the HEAT group and remained relatively unchanged for the NEUT condition ($P<0.05$).

Forehead $\dot{m}_{sw}$ decreased for both the NEUT and HEAT acclimation regimes during the resting and 30% $\dot{W}_{peak}$ periods. The change was significantly greater following NEUT acclimation ($P<0.05$). During the 45% $\dot{W}_{peak}$ period, $\dot{m}_{sw}$ increased in the HEAT condition ($P<0.05$). A significantly greater decrease in forehead $\dot{m}_{sw}$ was observed for the NEUT condition when data were analysed across the full exposure ($P<0.05$). The $\dot{m}_{sw}$ threshold for the forehead decreased for both the NEUT and HEAT conditions, while the $\dot{m}_{sw}$ sensitivity increased for both groups, however, the changes were not significant between conditions ($P>0.05$).

Forearm $\dot{m}_{sw}$ increased slightly for both acclimation groups over the total time period, however, the changes were not significant ($P>0.05$). No significant differences were apparent at each of the three HST phases for either condition. Forearm $\dot{m}_{sw}$ produced similar threshold and sensitivity responses to those displayed by the forehead ($P>0.05$). The sweat threshold decreased and sensitivity increased for both the NEUT and HEAT conditions, however, the changes were not significant between conditions ($P>0.05$). The change in mass scores between HSTs showed a slight decrease in mass lost, however, these were not significant between the NEUT and HEAT groups ($P>0.05$).

A comparison of the change scores between the two acclimation regimes, shows that both the NEUT and HEAT regimes produced acclimation effects. However, the magnitude of the changes were greater following the HEAT condition for $T_{ac}$, forehead

$\dot{m}_{sw}$, and $\overline{T}_{sk}$. While the pre-and post-acclimation results show that both protocols produce acclimation effects, the change values suggest that HEAT acclimation may be the most efficient in terms of the extent to which physiological adaptation occurs.

# CHAPTER FIVE: DISCUSSION

## 5.1 DISCUSSION

This project investigated adaptations to heat stress following extended exposure to two thermal environments. The purpose was to evaluate the importance of air temperature, and hence peripheral thermoreceptor input, in the acclimation process. This was achieved by keeping body $T_c$ (strain) constant across experimental conditions. Reduced physiological strain was observed for both experimental groups, however, despite maintaining isothermal strain, an acclimation effect, as defined by a significant decrease in $T_{ac}$, was only apparent for the HEAT group. The general trend was towards a greater decrease in physiological strain following HEAT acclimation, resulting in the following general adaptations: more prolific sweating, decreased $T_{ac}$, $f_c$, $\overline{T}_{sk}$, perceived exertion, and thermal sensation. Decreased physiological strain was portrayed by the NEUT group through decreased $f_c$ and thermal sensation.

### 5.1.1 Acclimation

The external work (cycling) required to elevate and maintain body $T_c$ was significantly greater for the group training in the neutral environment. The difference in external work employed for both groups was designed to ensure that the average time to reach the 1°C elevation in $T_{re}$ was about 30 minutes, regardless of the environmental temperature. $T_{re}$ was then maintained at these relative elevations for the remainder of the hour each day. A similar method of equating strain during acclimation was used by Havenith and van Middendorp (1986). However, they elevated $T_c$ to a standard of 38.3°C, which did not take into account the pre-exercise body $T_c$ for individual subjects. As a result, subjects were exposed to different levels of strain depending upon initial $T_c$, therefore, the relative importance of central thermal input was not adequately isolated by this design.

Both the NEUT and HEAT acclimation regimes produced increases in $\dot{V}_{O2peak}$

following the ten days of training. Researchers suggest that an increased $\dot{V}_{O2peak}$ improves physiological responses during exercise in the heat and hastens the acclimation process. Aerobic training that improves $\dot{V}_{O2peak}$, elevates $T_c$ and elicits sweating. Thus, thermoregulatory function and heat tolerance can be improved as a result of an improved sweating response and reduced cardiovascular strain in the heat (Nadel *et al.*, 1974; Henane *et al.*, 1977; Cadarette *et al*, 1984). The increases in $\dot{V}_{O2peak}$ observed in this experiment were not significant in either condition, suggesting that the increase in aerobic power did not account for the greater acclimation response seen in the HEAT group. This finding may be a result of the acclimation protocol, in that the workload for each subject was dependent on the elevation and maintenance of $T_c$ 1°C above their resting value. If the target $T_c$ was higher and both acclimation groups had to cycle at higher work rates to achieve this goal, there may have been a significant increase in $\dot{V}_{O2peak}$ and greater differences between groups.

Mean skin temperature was significantly lower for the NEUT group than the HEAT group, averaged over the ten day exposure. Since $\overline{T}_{sk}$ is a function of $T_A$ and SkBF, and observed to be independent of internal temperature across a wide temperature range (Nadel *et al.*, 1971a), the difference in $T_A$ between the two conditions caused this $\overline{T}_{sk}$ response. $\overline{T}_{sk}$ showed no significant differences from day one to day ten of acclimation in either of the two acclimation conditions. Both Roberts *et al.* (1977) and Avellini *et al.* (1980) found similar $\overline{T}_{sk}$ responses following ten days of training in neutral and hot environments. The general trend following acclimation, however, is that $\overline{T}_{sk}$ decreases as acclimation sessions progress (Shvartz *et al.*, 1979; Shapiro *et al.*, 1981; Havenith and van Middendorp, 1986). This change in skin temperature may result from physiological adjustments in sweat evaporation and cutaneous blood flow, which may increase at any given core and skin temperatures as acclimation progresses. Thus heat balance is achieved at lower core and skin temperatures during exercise in the heat.

Although acclimation $T_c$ values were slightly different between conditions, they were relatively close, and since the adaptations to heat stress were greater following the HEAT regime, the importance of exposing the skin to external thermal stress during the

acclimation process has been demonstrated. The role of skin temperature in determining $\dot{m}_{sw}$ has previously been investigated. Nadel *et al.* (1971a) revealed that by clamping $T_{skl}$ at different levels during skin heating, the $T_{skl}$ exerted a modifying effect on the output from the central controller, acting as a multiplier in the determination of local sweating rate. Chen and Elizondo (1974) investigated the differences between whole body and $T_{skl}$ effects in acclimation, as depicted by $\dot{m}_{sw}$ in a number of experiments. It was concluded that while an elevated $T_c$ was important to the improvement in sweat gland function, it must be in conjunction with a raised $T_{skl}$. The current data indicate that not only is an elevated $T_{skl}$ important to thermoregulatory function, but is important to the whole process of acclimation. While lower physiological strain was attained by the NEUT group in the present experiment, more complete adaptation was apparent in the HEAT group.

Reduced physiological strain was depicted by a significantly lower $f_c$ in the HEAT condition of 20 $b.min^{-1}$ from day one to day ten of the acclimation regime. Other studies have also reported decreases in $f_c$ following acclimation. Havenith and van Middendorp (1986) observed significant reductions in $f_c$ following heat acclimation amounting to 10 to 14 $b.min^{-1}$. Avellini *et al.* (1980) also found decreases in $f_c$ in the magnitude of 27 to 30 $b.min^{-1}$, following six days of prolonged dry heat exposure. Only a slight reduction in $f_c$ was observed following the NEUT condition. While $f_c$ was similar between the two groups by day ten of each exposure, the NEUT group had a significantly lower $f_c$ on day one of acclimation. This leads to the possibility that the NEUT group had greater training status than the HEAT group prior to acclimation, thus, only a small change in $f_c$.

### 5.1.2 Heat stress test one

$T_{ac}$ showed differing responses in accordance with the increasing demands placed on the subject as the HST progressed. Upon entering the chamber, $T_{ac}$ decreased. This was the result of the subject moving from the cooler environment of the anteroom, where $T_{ac}$ was stabilised, into the hot environment of the climate chamber. This can be explained by the occurrence of cutaneous vasodilation in response to the hot

environment (Rowell, 1974). More blood is shunted to the periphery so that heat delivered to the skin passes to the environment, and a smaller proportion is returned to the core. That is, venous blood is cooler than arterial blood, and $T_{ac}$ decreases during the early stages. However, as $\overline{T}_{sk}$ began to rise as a function of both $T_A$ and elevated SkBF, the temperature gradient between the environment and the skin, and the core and the skin, was reduced, causing $T_{ac}$ to stabilise during the resting phase of the HST. At the commencement of the first exercise phase (30% $\dot{W}_{peak}$), $T_{ac}$ increased rapidly. This was the result of an elevation in the rate of heat production with the initiation of exercise, which initially exceeds the rate of heat dissipation, causing a nett storage of body heat (Nadel, 1985). $T_{ac}$ stabilisation was also reached during this phase, since $T_{ac}$ was sufficiently raised beyond the thresholds for vasodilation and sweating, thus increasing heat transfer to the surface and its subsequent elimination (Kenney and Johnson, 1992). Heat dissipation now matched metabolic heat production but with $T_c$ shifted to a new, elevated steady state. Due to the increased metabolic demands imposed by the third HST phase (45% $\dot{W}_{peak}$), $T_{ac}$ again increased, but now with no evidence of stabilising. The metabolic and environmental demands imposed on the subject during this pre-acclimation heat exposure caused heat production to outweigh heat dissipation. Avellini *et al.* (1980) reported similar increases in $T_c$ without stabilising during a pre-acclimation HST in 36°C, as a result of similar demands placed on subjects.

The commencement of the pre-acclimation HST caused $\overline{T}_{sk}$ to rise rapidly in response to the hot environment. Following the rapid rise, $\overline{T}_{sk}$ continued to increase progressively before reaching an equilibrium state at a temperature close to the $T_A$ of the surroundings. This response is in accordance with the finding by Nadel *et al.*(1971a) that $\overline{T}_{sk}$ is primarily a function of the $T_A$. Avellini *et al.*(1980) found a similar pre-acclimation response in a 36°C environment, where $\overline{T}_{sk}$ increased rapidly until it was close to the $T_A$, and then plateaued for the remainder of the exposure.

Thermal sensation scores followed a similar pattern to $\overline{T}_{sk}$, and a relationship between these two variables has been reported in the literature. Hardy (1961) reported that thermal sensation is derived primarily from cutaneous thermoreceptors and the rate

of change in $\overline{T}_{sk}$. A high correlation between $\overline{T}_{sk}$ and thermal sensation (r=0.71) was observed by Kamon *et al.* (1974) while subjects performed work in different environments. Maw *et al.* (1993) also reported that thermal sensation scores generally followed the changes observed in $\overline{T}_{sk}$, in a study of the perceptual and physiological responses of subjects during steady state exercise in a range of environments.

Skin blood flow oscillated throughout the three phases of the pre-acclimation HST, particularly during the resting phase. This is a natural occurrence of the rhythmic activity in the cutaneous sympathetic nerves (Johnson, 1986). Vasodilation in the forearm was evident in the hot environment. Rowell (1974) attributes vasodilation, as a result of direct whole body heating, to an almost immediate increase in $f_c$ and cardiac output, which is directed to the skin with further supplementation by the reduction in splanchnic and renal blood flow. The commencement of the 30% $\dot{W}_{peak}$ exercise phase showed an initial decrease in SkBF, which may be the result of exercise onset vasoconstriction. Kellogg *et al.* (1991) reported that the cutaneous vasoconstriction elicited by the initiation of exercise was solely the result of increased active vasoconstrictor tone, since reductions in cutaneous vascular conductance were abolished with the initiation of exercise by bretylium treatment in both normothermia and heat stress. However, SkBF to the forearm is controlled by dual efferent mechanisms including the noradrenergic active vasoconstrictor system, which is responsible for exercise onset vasoconstriction, and an active vasodilator system (Johnson, 1986). Thus, once $T_c$ was elevated sufficiently, active vasodilation increased SkBF slightly, in conjunction with increasing $T_{ac}$ before reaching a plateau during this phase. During the 45% $\dot{W}_{peak}$ exercise phase, SkBF increased rapidly to a new plateau level. This response is in accordance with the literature in which SkBF portrays a characteristic internal temperature threshold beyond which it rises with increasing $T_c$ (Johnson and Park, 1981). The slight reduction in SkBF towards the end of the 45% $\dot{W}_{peak}$ exercise phase, may reflect excessive cardiovascular strain, as a result of maximal vasodilation causing reduced venous return, stimulating the cutaneous vasoconstrictor drive so that blood flow can be diverted from the periphery to maintain blood pressure (Rowell, 1974).

During a substantial period of the resting phase of the HST, heat dissipation achieved through evaporative loss was minimal. However, as the core to skin temperature gradient was reduced, sweating was initiated to dissipate the heat load. With the introduction of the 30% $\dot{W}_{peak}$ exercise phase, $\dot{m}_{sw}$ increased rapidly at the forehead and forearm, to approximately 1.6 and 1.2 $mg.cm^{-2}.min^{-1}$ respectively, to accommodate for the production of metabolic heat, and in an attempt to retain thermal equilibrium (Nadel, 1979). Forehead $\dot{m}_{sw}$ remained greater than forearm $\dot{m}_{sw}$ for the remainder of the HST. The initiation of the 45% $\dot{W}_{peak}$ exercise phase produced a further rapid increase in $\dot{m}_{sw}$ to approximately 2.2 $mg.cm^{-2}.min^{-1}$ for the forehead and 1.6 $mg.cm^{-2}.min^{-1}$ for the forearm. The greater $\dot{m}_{sw}$ produced by the forehead has been reported earlier by Hertzman *et al.* (1952), who exposed resting subjects to environmental temperatures in the range of 24°C to 38°C. It was shown that forehead $\dot{m}_{sw}$ was much higher than forearm $\dot{m}_{sw}$ across the temperature range. The difference in $\dot{m}_{sw}$ produced by the two regions supports the work of Nadel *et al.*(1971b), where it was demonstrated that individual regions of the skin have different sweating responses for a given $T_c$, the forearm having the lowest sweat output of five different body regions.

During the pre-acclimation HST, $\dot{m}_{sw}$ threshold core temperatures varied from 36.5°C to 39.2°C for both the forehead and the forearm. The mean threshold temperatures of 37.6°C for the forehead and 37.3°C for the forearm, derived prior to acclimation, were similar to data observed in the literature. Both Nadel *et al.* (1974) and Avellini *et al.* (1982) reported a pre-acclimation mean sweating threshold of 37.4°C. $\dot{m}_{sw}$ sensitivity also varied from 0.6 to 4.4 $mg.cm^{-2}.min^{-1}.°C^{-1}$ for the forehead and 1.0 and 3.9 $mg.cm^{-2}.min^{-1}.°C^{-1}$ for the forearm. The mean $\dot{m}_{sw}$ sensitivities were 2.7 and 1.9 $mg.cm^{-2}.min^{-1}.°C^{-1}$ for the forehead and forearm respectively, however, these values were higher than those recorded in the literature prior to acclimation. A mean sensitivity value of 1.1 $mg.cm^{-2}.min^{-1}.°C^{-1}$ was recorded by Roberts *et al.* (1977) and Avellini *et al.* (1982). Since testing was conducted during winter, reducing the effects of natural acclimatisation, the greater sensitivities reported in the present study may be the result of subjects having greater training status prior to acclimation. The mean $\dot{V}_{O2peak}$ values of subjects in this study were considerably higher than those of

subjects in previously reported studies (Roberts *et al.*, 1977; Pandolf *et al.*, 1977). The differences in threshold sweat rates and sensitivities between the present study and the literature may also be due to methodological differences during the pre-acclimation HSTs, with the most distinct difference being the positioning of the $\dot{m}_{sw}$ capsule. In the studies by Nadel *et al.* (1974), Roberts *et al.* (1977) and Avellini *et al.* (1982), $\dot{m}_{sw}$ was measured from the chest, and since $\dot{m}_{sw}$ shows different regional responses (Nadel *et al.*, 1971b), comparisons to the forehead and forearm may not be valid. Comparisons to the literature may also be difficult since the pre-exposure tests were conducted in a much cooler environment than that employed in the present study.

The response shown by $f_c$ during the pre-acclimation HST in the present study, shows the typical response patterns to heat and exercise. At the commencement of both exercise phases, $f_c$ increased sharply to new plateau levels in response to the changes in exercise intensity in the heat. Avellini *et al.* (1980) found similar $f_c$ responses to exercise in the heat, prior to acclimation. $f_c$ at the conclusion of the first HST was approximately 185-190 $b.min^{-1}$ indicating marked cardiovascular strain. Senay *et al.* (1976) suggested that $f_c$ increases in an attempt to maintain blood pressure and cardiac output as a result of maximal vasodilation causing reduced venous return (lower cardiac filling pressure and lower stroke volume). While perceived exertion scores are related to $f_c$ by a factor of ten (Borg, 1961), the scores reported during the pre-acclimation HST remained lower than $f_c$. Maw et al. (1993) found the factor of ten relationship to be changed in a hot environment, since the $f_c$ at the same RPE score was elevated in the heat compared to a cool condition. As a result, it was suggested that perceived exertion may be confounded when the cardiac stimulus includes a significant thermal drive, in addition to that provided by exercise.

### 5.1.3 Pre- versus post-acclimation heat strain

Reduced physiological strain in the HEAT group was demonstrated by significantly lower $T_{ac}$, during all phases of the second HST. Havenith and van Middendorp (1986) found a similar reduction of 0.2°C for $T_{re}$ and 0.3°C for oesophageal temperature ($T_{oe}$), following seven days of heat acclimation using a similar

isothermal (controlled hyperthermia) protocol. Significant decreases in $T_c$ are standard findings in other studies following heat acclimation protocols (Pandolf *et al.*,1977; Houmard *et al.*, 1990; Mitchell *et al.*, 1976), and it is this change that was used to define the presence of an acclimation effect. However, no significant change in $T_{ac}$ was apparent following the NEUT acclimation protocol in this study. A similar result was found by Shvartz *et al.*(1973) in a comparison study of three acclimation protocols in different thermal environments. They found that $T_{re}$ was not reduced following six days of acclimation in a neutral environment, as compared to acclimation in hot wet and hot dry environments. One possibility for the absence of a reduction in $T_{ac}$ in the NEUT group may be that the acclimation protocol was neither of sufficient intensity or duration. In the present study the stimulus was only ten days. Gisolfi and Robinson (1969) had subjects undertake six weeks of intensive interval training that elevated $T_{re}$ to approximately 39.7°C and found that post-acclimation $T_{re}$ decreased markedly as a result of the extended exposure. Avellini *et al.*(1982) found similar reductions in $T_{re}$ in subjects participating in four weeks of intensive land training. A decrease in $T_{ac}$ may have occurred in the NEUT group in the present study if the intensity and duration of the protocol was greater.

Steady state forearm SkBF was not significantly different following either the NEUT or HEAT acclimation regimes. Whether SkBF increases or decreases following acclimation varies within the literature. Wood and Bass (1960) found no overall change in forearm SkBF, using plethysmographic determinations, between the beginning and end of two heat acclimation protocols of six and nine days respectively. However, during the third and forth days of acclimation, SkBF reached minimum values, coinciding with the disappearance of heat strain symptoms. It was suggested that the peripheral vasomotor responses observed were important to the early stages of acclimation and then possibly masked by other adjustments, since acclimation was maintained for the remaining days of the protocol. Possible early adaptations in SkBF could not be detected in the present study since measurements were only recorded prior to, and following acclimation. Hellon and Lind (1955) reported reduced forearm SkBF following passive acclimation, where subjects were exposed to humid heat for two hours daily for nine consecutive days. In contrast, Fox *et al.*(1963) reported that both

forearm and hand blood flow was higher following an acclimatisation regime where $T_c$ was passively elevated to approximately 38.5°C on successive days. In a study where exercise was used as the stimulus for the increase in $T_c$, Havenith and van Middendorp (1986) reported slight reductions in forearm SkBF following a seven-day heat acclimation protocol, and suggested that since forearm SkBF seemed reduced, the $T_c$:$\overline{T}_{sk}$ gradient would be increased. However, since the gradient remained equal, Havenith and van Middendorp (1986) suggested that SkBF in other parts of the body may not have decreased. Based on this assumption, the present study indicates that SkBF has changed in other body regions since the mean $T_c$:$\overline{T}_{sk}$ gradient increased from 0.1°C to 0.4°C in the HEAT group, and decreased from 0.4°C to 0.1°C in the NEUT group following acclimation.

It is not known how well plethysmographic determinations at the extremities of the limbs represent SkBF in other regions of the body. In the present study, SkBF was measured at various body regions during the final five minutes of each of the three phases of the HST. There was a significant decrease in SkBF to the forehead during the last phase of the HST following the HEAT condition. A significant decrease was also found in the upper back during the 30% $\dot{W}_{peak}$ phase following the NEUT acclimation. No significant differences were found in the chest, upper back, upper arm or thigh regions following the HEAT condition, or the forehead, chest, upper arm or thigh regions following the NEUT condition. In a comparison study of blood flow response of the forearm and calf to thermal stress during dynamic exercise, Nishiyasu *et al.* (1992) found blood flow differences between the two regions and suggested that the SkBF response may not be uniform over the entire body surface. Similar SkBF variability was observed in the current study (Figure 4.6), although these differences remained uniform within and between the HSTs. The findings in the present study imply that forearm SkBF may not effectively demonstrate SkBF responses in other body regions. Thus, while SkBF may be reduced, its observation may well be dependent upon the site chosen to study SkBF.

The HEAT condition produced significantly lower $\overline{T}_{sk}$ during all phases of the second HST. This response was also found in the study by Havenith and van

Middendorp (1986) in which $\overline{T}_{sk}$ was significantly lower following the acclimation procedure by approximately 0.3°C. Similar results of lower $\overline{T}_{sk}$ following heat acclimation have been reported by others (Pandolf *et al.*, 1977; Shvartz *et al.*, 1973; Shvartz *et al.*, 1979). Following NEUT training, however, $\overline{T}_{sk}$ was found to be significantly higher. It was thought that the increase incurred by the NEUT group may have resulted from differences in the $T_A$ between the pre- and post-HST, since $\overline{T}_{sk}$ has been demonstrated to be a function of $T_A$ (Nielsen and Nielsen, 1965). However, since mean $T_A$ was 39.8°C and 39.7°C in the pre- and post-acclimation HST respectively, and was not significantly different between tests, the increase in $\overline{T}_{sk}$ must be due to changes in SkBF and $\dot{m}_{sw}$. While changes in SkBF were not significant following NEUT acclimation, except for a reduction in SkBF in the upper back, it was observed to be greater at the forehead, chest and thigh during the 30% $\dot{W}_{peak}$ exercise phase of the second HST, which may have contributed to the increase in $\overline{T}_{sk}$. Forearm $\dot{m}_{sw}$ increased during the second HST for the NEUT group, however, it was not as great as the decrease in forehead $\dot{m}_{sw}$. Thus, cooling efficiency may have decreased, contributing to the increase in $\overline{T}_{sk}$.

In relation to $\overline{T}_{sk}$, thermal sensation was found to be significantly lower following both acclimation regimes, the change being more pronounced for the HEAT group. Since $\overline{T}_{sk}$ was significantly lower following HEAT acclimation, the greater decrease in thermal sensation for this condition may have been derived from the greater heat stimulus on the peripheral thermoreceptors (Hardy, 1961). A relationship between thermal sensation and $\overline{T}_{sk}$ was reported by Pandolf *et al.* (1977) who found a significant decrease in thermal sensation following nine days of heat acclimation in conjunction with a significant decrease in $\overline{T}_{sk}$.

Reduced cardiovascular strain was shown by significantly lower $f_c$ following both the NEUT and HEAT conditions across all phases of the HST. Similar observations have been made in other studies following acclimation regimes (Mitchell *et al.*, 1976; Shapiro *et al.*, 1981; Cadarette *et al.*, 1984). Rowell *et al.* (1967) assumed that decreases in $T_c$ and $\overline{T}_{sk}$ lead directly to decreases in $f_c$. However, since $T_c$ did not change, and $\overline{T}_{sk}$ increased following NEUT acclimation, the changes may have been the

result of increased plasma volume. Such a result has been reported by Senay *et al.* (1976), where it was found that an increase in plasma volume was significantly related to decreases in $f_c$ and increases in stroke volume, during the early days of exposure to an acclimation regime.

While the decreases in $f_c$ were similar following both acclimation regimes, RPE scores, differentiated into three different scores for the whole body, chest and legs (Pandolf, 1978) were only significantly lower following HEAT acclimation. Since $f_c$ adjustments were similar between the two groups following acclimation, and $T_c$ was relatively close between conditions, RPE may be related to the decrease in $\overline{T}_{sk}$ in the HEAT group. Maw *et al.* (1993) suggested that $\overline{T}_{sk}$, rather than $T_c$ was a contributor to effort perception, thus RPE may be more sensitive to peripheral input. In a study comparing young and old subjects during heat acclimation Pandolf *et al.* (1988), it was found that RPE was generally higher for the young subjects. In a comparison of the changes in $f_c$ between the two groups, no differences were found and it was suggested that the different RPE values must be associated with other sensory cues. This study also supports the possibility that differences in $\overline{T}_{sk}$ may provide the stimulus for changes in RPE since $\overline{T}_{sk}$ was consistently lower for the older subjects throughout the acclimation protocol.

Adjustments to $\dot{m}_{sw}$ were produced by acclimation in both the NEUT and HEAT conditions. During the resting and 30% $\dot{W}_{peak}$ exercise phase of the second HST, forehead $\dot{m}_{sw}$ was equivalent to pre-acclimation values in the HEAT group, indicating a more sensitive sweating response in the presence of lower $T_{ac}$. During the final 20 minutes of the HST, the HEAT group produced significantly greater $\dot{m}_{sw}$ than pre-acclimation levels, while $T_{ac}$ was also lower. It is probable that $T_{ac}$ was held lower by this greater $\dot{m}_{sw}$, which facilitated heat loss, but with some physiological cost. The general increase in the sweating response following heat acclimation has previously been demonstrated in the literature (Libert *et al.*, 1983; Sato *et al.*, 1990). In contrast, the first two phases of the second HST showed lower forehead $\dot{m}_{sw}$ for the NEUT group, indicating elevated sweating efficiency in the presence of equal $T_{ac}$ in the pre- and post-acclimation HSTs. Havenith and van Middendorp (1986) found a similar

sweating response following a controlled $T_c$ acclimation regime, where sweat production was slightly reduced in the presence of a lower $T_c$, suggesting increased sweating efficiency. Thus, lower $\dot{m}_{sw}$ is required to maintain a similar or lower $T_c$, implying a more efficient system. While an increase in $\dot{m}_{sw}$ to maintain lower $T_c$, as depicted by the HEAT group, suggests improved heat tolerance, this adjustment may prove detrimental to long term exposure due to greater losses of fluid and electrolytes, and eventually, greater heat strain. The maintenance of $T_c$ with the production of less sweat following acclimation implicates a more practical adjustment, especially in relation to long term exposures.

Forearm $\dot{m}_{sw}$ increased during the final phase of the post-acclimation HST in both acclimation groups and during the 30% $\dot{W}_{peak}$ exercise phase for the HEAT condition. Based on the findings for the $\dot{m}_{sw}$ adaptations for the forehead and forearm, the third hypothesis can be partially accepted, in that the sudomotor steady state response was greater following HEAT acclimation during a standard thermal stress. Since total body weight losses during the first and second HST's were not significantly different following either acclimation regime, and increases in the forearm $\dot{m}_{sw}$ were relatively greater than the forehead following acclimation, a possible redistribution of sweat may have occurred from the forehead to the forearm, or from central to peripheral skin regions. A greater production of sweat on the limbs following acclimation has been reported in the literature. Shvartz *et al.* (1979) in a study of the effects of eight days of heat acclimation and conditioning on three different sweating regions on the body, found a larger increase in limb $\dot{m}_{sw}$ (forearm and thigh) than trunk $\dot{m}_{sw}$ following the acclimation regimes. According to Collins *et al.* (1965), the sweating intensity becomes more uniform over the skin surface following acclimation, and since the sweat glands of the forearm are more densely distributed than those of the trunk, the potential for increasing output per unit area is greater.

The effects on the sweating threshold and sensitivity found following both acclimation regimes were not significant. On the basis of these results, the first and second hypotheses, where it was stated that both the NEUT and HEAT acclimation regimes would produce equivalent decreases in $T_c$ thresholds and increases in sweating

sensitivity, cannot be accepted. However, the general trend showed that both groups increased sweating sensitivity and decreased the threshold $T_c$ for sweating. The HEAT group also showed decreased $\overline{T}_{sk}$ at the sweating threshold, which was not evident following the NEUT condition. These changes favoured the HEAT trained subjects indicating greater adaptation. As a result of ten consecutive days of physical training, followed by ten days of heat acclimation, Nadel *et al.* (1974) reported a greater increase in the sensitivity of the sweating response in relation to $T_c$ following physical training, while heat acclimation showed a greater reduction in the threshold $T_c$ for sweating. However, while these adjustments were made, it appears that the assumption made by Nadel *et al.* (1974) that physical training produced peripheral adaptations and heat acclimation produced central adaptations, may not be quite so distinct. In a study with a similar experimental design, Roberts *et al.* (1977) found a small increase in the sensitivity of the sweating relation and a reduction in the threshold for sweating following physical training. Following heat acclimation, a large reduction in the sweating threshold was evident without any further increase in sweat sensitivity. It is possible that both the central and peripheral adaptations would have been greater following heat acclimation alone in the above studies, if the subjects had not been exposed to the physical training protocol first. In a study where the effects of heat acclimation were studied in isolation, using a controlled $T_c$ technique (Havenith and van Middendorp, 1986), as was evident in the present experiment, both central and peripheral sudomotor adaptations were reported through the lowering of the threshold for sweating and increasing sensitivity. It seems that the possible mechanisms for $\dot{m}_{sw}$ adjustments in different acclimation environments are still yet to be isolated.

### 5.1.4 Comparisons between acclimation protocols.

It is well documented that both artificial heat acclimation and physical training which elevates $T_c$, generally improves the heat tolerance of an individual (Pandolf, 1979; Rowell, 1974; Avellini *et al.*, 1982). However, few groups have attempted to equate physiological strain between these two methods of acclimation, making comparisons difficult between the acclimation states induced by these means. Since $T_c$ was controlled between conditions in the present study, equating physiological strain,

comparisons could be made between the efficiency of combined heat and exercise acclimation compared with physical training in a neutral environment.

Both $T_{ac}$ and $\overline{T}_{sk}$ showed significantly greater reductions of 0.4°C and 0.6°C respectively, following the HEAT acclimation regime. In contrast, the NEUT group showed relatively no change in $T_{ac}$ and a slight increase in $\overline{T}_{sk}$. A similar result was found following heat acclimation in a study comparing three different acclimation regimes. Shvartz *et al.* (1973) reported that the greatest reductions in $T_{ac}$ and $\overline{T}_{sk}$ were found following acclimation in a hot dry environment, and suggested that these adjustments contributed to the transfer of a substantial amount of heat to the periphery, contributing to greater thermal stability. Pronounced reductions in these physiological variables have been found in other studies, following heat acclimation (Wells, 1980; Avellini *et al.*, 1982). Thermal sensation decreased following both conditions, however, the change was significantly greater following the HEAT acclimation regime, corresponding to changes in $\overline{T}_{sk}$ (Pandolf *et al.*, 1979).

Documentation of the comparison between changes in SkBF following acclimation in the two different thermal environments is limited, and has not been attempted using the LDV technique. The present study showed virtually no change in forearm SkBF following either NEUT or HEAT acclimation. The pattern portrayed by the literature is not consistent as to whether SkBF decreases (Hellon and Lind, 1955), increases (Fox *et al.*, 1963), or does not change (Wood and Bass, 1960) as a result of acclimation. However, while Wood and Bass (1960) found no overall change in SkBF, consistent with the present study, SkBF was shown to be reduced during the third and forth days of an acclimation regime that coincided with the disappearance of heat strain symptoms. The possibility of such an adjustment may have gone undetected in the present study.

While $f_c$ portrayed decreases of 8.2 and 8.6 $b.min^{-1}$ following the NEUT and HEAT conditions respectively, the changes were not significant between groups. This observation indicates that both acclimation regimes produced similar reductions in cardiovascular strain. Shvartz *et al.* (1979) also found decreases in $f_c$ following

acclimation regimes in hot and neutral environments, with the decrease being much greater for the heat acclimated group. However, this may have been the result of different levels of physiological strain imposed on the two groups during acclimation, since both groups followed the same exercise protocol, regardless of the environmental temperature.

Perceived exertion decreased following the HEAT acclimation regime only. The change in $f_c$ was not significant between groups, therefore, the difference in RPE scores may be related to other sensory cues. In an acclimation study by Pandolf *et al.* (1988), $\overline{T}_{sk}$ was lower in one group throughout the duration of the regime, corresponding with lower RPE scores. Since the difference in $\overline{T}_{sk}$ is the only other distinct change between the two conditions in the present study, this variable may have provided the stimulus for the decrease in RPE.

During the 45% $\dot{W}_{peak}$ phase of the post-acclimation HST, forehead $\dot{m}_{sw}$ increased in the HEAT condition. This adjustment was significantly greater than the decrease in forehead $\dot{m}_{sw}$ in the NEUT group. Pandolf *et al.* (1977) reported an enhanced sweating response following heat acclimation of subjects. Forearm $\dot{m}_{sw}$ increased in both groups but the changes were not significant between groups. This suggests that both groups showed similar increases in $\dot{m}_{sw}$ distally, and appears to indicate that the peripheral shift in $\dot{m}_{sw}$ was not dependent upon $\overline{T}_{sk}$ during acclimation, but was $T_c$ dependent. The increase in $\dot{m}_{sw}$ at the distal regions during both heat and neutral acclimation regimes has also been reported by Shvartz *et al.* (1979), although $T_c$ was not equated between groups. $\dot{m}_{sw}$ thresholds were decreased and sensitivity increased following both NEUT and HEAT acclimation at the forehead and forearm, however, the changes were not significant between the two conditions. In the study by Nadel *et al.* (1974) the change in sweating sensitivity was greater following neutral training, and the change in threshold was greater following heat acclimation. However, as indicated by the present study, whether the change following NEUT or HEAT acclimation can be distinguished as being either central of peripheral in origin, is still not clear.

As a result of the comparisons made on the magnitude of changes between the NEUT and HEAT acclimation regimes, it is shown that the reduction in physiological strain was greater following HEAT acclimation. The HEAT group demonstrated reduced thermal strain due to: decreased $T_{ac}$, $f_c$, $\overline{T}_{sk}$, perceived exertion and thermal sensation. Sweating was more prolific, particularly during the final phase of the HST for the forehead and both exercise phases for the forearm. This finding provides partial support for the third hypothesis, in that the steady state sudomotor response improved following both acclimation protocols but was greater following HEAT acclimation. On the basis of the results obtained, the peripheral thermoreceptors are shown to play an integral role in the acclimation process.

## 5.2 GENERAL CONCLUSIONS

Both artificial heat acclimation and physical training that elevates $T_c$, generally improve heat tolerance. However, little is known about the mechanism underlying this process. Since it is known that both core and peripheral temperatures provide critical input to the thermoregulatory process, an attempt was made to differentiate between the roles of these temperature inputs by keeping $T_c$ constant across acclimation conditions. Thus the importance of skin temperature during the acclimation process in optimising heat tolerance could be determined.

Following isothermal (controlled hyperthermia) acclimation regimes in both neutral and hot environments, greater physiological adaptations were observed in the HEAT acclimated group due to decreased $T_{ac}$, $f_c$, $\overline{T}_{sk}$, perceived exertion and thermal sensation, and increased $\dot{m}_{sw}$. The NEUT group did show adaptation through decreased $f_c$, thermal sensation, and an increased sweating efficiency as a result of decreased $\dot{m}_{sw}$ for an equal $T_c$. While no changes in steady state SkBF were apparent, these findings provide partial support for the third hypothesis in that the sudomotor steady state responses were greater following HEAT rather than NEUT acclimation. The first and second hypotheses could not be accepted since the $\dot{m}_{sw}$ thresholds and sensitivities did not change significantly between groups.

It must be noted, however, that although subjects were matched prior to data collection, pre-exposure differences in acclimation state of the two subject groups became apparent. Analyses of the pre-acclimation HST data revealed that $T_{ac}$, $f_c$, $\overline{T}_{sk}$ were lower in the NEUT group during this test than the HEAT group. Forehead and forearm $\dot{m}_{sw}$ were greater for the NEUT group. These results suggest that the NEUT group had a higher state of natural acclimatisation than the HEAT group prior to the acclimation regimes. As a result, comparisons between pre- and post-acclimation regimes may have been confounded, since subjects in the NEUT group portrayed less physiological strain prior to acclimation, and each group acted as their own control. If acclimation status had been more closely controlled, comparisons between the two groups may have revealed greater acclimation differences.

Despite the possible difference in acclimation states prior to the commencement of testing, both the NEUT and HEAT groups produced elevated $\dot{m}_{sw}$ at the distal body regions and showed similar decreases in $f_c$. These findings appear to indicate that these adaptations were dependent upon $T_c$ rather than an elevated $\overline{T}_{sk}$ during the acclimation regime. However, based on the majority of the adaptations, it seems that in accordance with the literature, the elevation of $\overline{T}_{sk}$, in conjunction with elevated $T_c$ during acclimation exposures verifies its importance in optimising physiological adaptation.

## 5.3 RECOMMENDATIONS FOR FUTURE RESEARCH

Although the present study provides verification of the importance of elevating $\overline{T}_{sk}$ during the acclimation process, the underlying mechanism remains unclear. While it is possible to equate thermal strain by means of maintaining constant $T_c$, further studies must be more attentive to subject matching. Prior to the acclimation regime, subjects should undergo a screening test to define current acclimatisation status. Havenith and van Middendorp (1990) defined current acclimatisation as the relationship between $\dot{m}_{sw}$ and $T_c$ in a hot dry environment, during a test that required subjects to work for three 30 minute periods at different percentages of $\dot{V}_{O2peak}$. A linear regression line was constructed from the $\dot{m}_{sw}$ equilibrium values at the three different steady state levels, from which the slope of the relationship and the intercept of $T_c$ for a $\dot{m}_{sw}$ equal

to zero could be determined. This method of determining the individual acclimatisation status of subjects could be utilised in future acclimation studies of this nature, so that groups may be more closely matched.

Further studies should also attempt to collect sudomotor and cutaneous vasomotor data throughout the acclimation exposures, since important adaptations may have occurred during this time. Such adaptations were evident during the study by Wood and Bass (1960), where SkBF changes occurred during the third and forth days of an acclimation regime but disappeared by the conclusion of the regime. In addition, data collection should be designed so that the vasomotor thresholds and gains can be established prior to, and following acclimation. This was not possible during the present study due to the nature of testing. More SkBF data needs to be collected from different body regions since it has been implied that forearm SkBF may not represent SkBF in other regions of the body very well.

While the importance of an elevated skin temperature during the acclimation process has been verified by equating $T_c$, further research should involve more precise control of this variable, possibly using a water perfused suit that can control skin temperature of the body. The ability to manipulate skin temperature using such apparatus, while maintaining a constant $T_c$, may provide more insight into this mechanism underlying improvements in heat tolerance.

Since it has been shown that the sweating response differs according to the body segment being measured (Nadel *et al.*, 1971b; Shvartz *et al.*, 1979), future research should measure $\dot{m}_{sw}$ at more body sites. Comparisons could also be made to $T_{skl}$ in response to different thermal environments. This would allow for a more detailed account of the adjustments to sweating recruitment patterns following acclimation regimes.

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# APPENDICES

## Appendix A

### A method for the determination of peak oxygen consumption

This pilot study was conducted to find the most suitable method for determining peak oxygen consumption ($\dot{V}_{O2peak}$) using a ramp cycle protocol, which could be used as the standard method for all experiments conducted in the laboratory.

Seven subjects were recruited; three male and four female elite middle to long distance runners (state and national level). Data for the determination of $\dot{V}_{O2peak}$ were collected on a breath-by-breath basis using a Quinton Q-Plex I system (Quinton Instrument Company, Q-Plex I, Seattle) that was comprised of a pneumotachograph (Hans Rudolph Inc., model 3813, Kansas City, U.S.A.), a zirconia oxide oxygen analyser, and an infrared absorption carbon dioxide analyser. Subjects breathed through a Hans Rudolph two-way valve (dead space 115 ml), connected to the Q-Plex I by 35mm of low resistance tubing. A headset held the valve in position while allowing freedom of movement. Subjects cycled on an electronically braked cycle ergometer (Lode Excalibur Sport, Netherlands). The seat and handlebars were adjusted in the vertical and horizontal planes for subject comfort.

The $\dot{V}_{O2peak}$ of each subject was elicited using a ramp cycle protocol, with work rate increments of 3 watts every 5 seconds, beginning at 0 watts (ambient temperature of 22°C). Each test was terminated at volitional fatigue, with a reduced work rate to allow recovery. Verbal encouragement was given to each subject.

*Post hoc* determination of $\dot{V}_{O2peak}$ was attempted with the following combinations of breath and time based averaging:

(i) the last 15, 20 and 30 second averaging of $\dot{V}_{O2}$ data;

(ii) the last 3, 5, 6, 9, 10 and 15 breaths were averaged; and

(iii) every 15 second interval over the entire test duration.

Averaging of the $\dot{V}_{O2peak}$ data over the nine different combinations produced similar values for each individual subject (Table A). Computation of the mean value for all subjects at each combination revealed $\dot{V}_{O2peak}$ data ranging from 4.15 to 4.20 $l.min^{-1}$, with no significant differences between methods ($P>0.05$).

On the basis of this study, $\dot{V}_{O2peak}$ was determined from the average of the oxygen consumption data over the last 15 seconds of the test. If the individual displayed a plateau in their data and they still continued the test until volitional fatigue, the $\dot{V}_{O2peak}$ score was then taken as the highest score on the data graph, provided it was not followed by a further 150 ml upward or downward fluctuation.

**Table A:** Peak oxygen consumption ($l.min^{-1}$ STPD) for individual subjects averaged over nine different combinations of time and number of breaths.

| Sub | last 15 sec. | last 30 sec. | last 20 sec. | 3 br. | 6 br. | 9 br. | 5 br. | 10 br. | 15 br. |
|---|---|---|---|---|---|---|---|---|---|
| F1 | 3.19 | 3.22 | 3.23 | 3.13 | 3.11 | 3.16 | 3.10 | 3.17 | 3.01 |
| M1 | 6.14 | 6.03 | 6.12 | 6.13 | 6.15 | 6.14 | 6.17 | 6.15 | 6.15 |
| F2 | 2.90 | 2.92 | 2.19 | 2.88 | 2.91 | 2.92 | 2.90 | 2.90 | 2.91 |
| F3 | 3.80 | 3.77 | 3.77 | 4.06 | 3.80 | 3.77 | 4.03 | 3.79 | 3.77 |
| F4 | 3.58 | 3.55 | 3.50 | 3.63 | 3.59 | 3.59 | 3.61 | 3.58 | 3.58 |
| M2 | 4.98 | 5.11 | 5.02 | 4.32 | 4.71 | 4.86 | 4.63 | 4.90 | 4.99 |
| M3 | 4.88 | 4.86 | 4.85 | 4.87 | 5.04 | 4.86 | 4.96 | 4.90 | 4.87 |
| $\overline{X}$ | 4.21 | 4.21 | 4.20 | 4.15 | 4.19 | 4.19 | 4.20 | 4.20 | 4.18 |
| s.d. | 1.13 | 1.11 | 1.10 | 1.09 | 1.09 | 1.09 | 1.09 | 1.08 | 1.09 |
| SEM | 0.41 | 0.42 | 0.42 | 0.41 | 0.41 | 0.41 | 0.41 | 0.41 | 0.41 |

Abbreviations: Sub= subject; M= male; F= female; $\overline{X}$= mean; s.d.= standard deviation; SEM= standard error of the mean.

## Appendix B

**Determination of a sufficient elevation in core temperature for an acclimation protocol using an isothermal strain technique.**

This pilot study was conducted to evaluate the magnitude of the rise in $T_c$ which might be encountered during a typical endurance training stimulus, and to determine whether a 1°C elevation in body core temperature was a sufficient stimulus during the acclimation protocols.

Five trained runners (22-40 years) were recruited for the pilot study. Rectal temperature was measured using a thermistor (Yellow Springs Instrument Co., Inc., YSI probe no. 401, Ohio), positioned 12cm beyond the anal sphincter. Data was recorded manually from a tele-thermometer (Yellow Springs Instrument Co., YSI model 46, Ohio). Runners monitored their $T_{re}$ immediately prior to, and following a normal running training session. Data recorded included; resting $T_{re}$, post-run $T_{re}$, the duration and intensity (cardiac frequency; $f_c$) of the run, and the ambient temperature ($T_A$).

The duration and intensity of the training sessions, and the $T_A$ varied between subjects (Table B). While the runners $T_c$ elevations ranged from 1.4°C to 1.9°C, depending on the intensity and duration of the session, the mean $T_{re}$ elevation was 1.7°C.

On the basis of the evaluation on the magnitude of the rise in $T_{re}$ it was decided that a 1°C elevation in $T_{re}$ during acclimation would be a sufficient stimulus for training in the artificial environment.

**Table B:** Evaluation of the magnitude of the rise in core temperature during a typical training session for individual subjects.

| Sub | Duration (min) | $f_c$ ($b.min^{-1}$) | $T_A$ (°C) | $T_{re}$ (°C) rest | $T_{re}$ (°C) post-run | $T_{re}$ (°C) diff |
|---|---|---|---|---|---|---|
| 1 | 40 | 150 | 22 | 37.0 | 38.9 | 1.9 |
| 2 | 60 | 160 | 18 | 37.2 | 38.6 | 1.4 |
| 3 | 90 | 160 | 18 | 37.5 | 39.2 | 1.7 |
| 4 | 50 | 165 | 15 | 37.0 | 38.7 | 1.7 |
| 5 | 45 | 170 | 17 | 36.9 | 38.5 | 1.6 |
| Mean | 57 | 161 | 18 | 37.1 | 38.8 | 1.7 |
| s.d | 19.9 | 7.4 | 2.5 | 0.2 | 0.3 | 0.2 |

Abbreviations: Sub= subject; $T_A$= ambient temperature; $T_{re}$= rectal temperature; diff= difference; s.d.= standard deviation.

## Appendix C

**Determination of the workload required to elicit a 1°C core temperature elevation from resting during acclimation sessions in hot and neutral environments.**

Since an isothermal (controlled hyperthermia) strain technique is not a common technique used in acclimation studies, this pilot study was conducted to determine the initial workload required to elevate $T_{re}$ 1°C within 30 minutes of acclimation sessions in both hot and neutral environments.

Five male subjects (aged 24.0 years ± 3.1; mass 78.6 kg ± 7.8; exclusive of the acclimation sample) participated in trials in both the neutral (22°C, RH 40%) and heat (38°C, RH 40%) environments to be used during acclimation. Rectal temperature ($T_{re}$) was measured using a thermistor (Yellow Springs Instrument Co., Inc., YSI probe no. 401, Ohio), positioned 12cm beyond the anal sphincter. Data was recorded manually from a tele-thermometer (Yellow Springs Instrument Co., YSI model 46, Ohio). Cardiac frequency ($f_c$) was recorded using a SportTester (Polar Electro, model PE 3000, Finland). Initially, the subject's age was taken to determine 75% of the predicted maximal $f_c$ using Karvonen's formula (Lamb, 1984). This was the intensity chosen for subjects to work at initially. Prior to testing, body mass was determined, subjects inserted the $T_{re}$ to a depth of 12 cm and were then fitted with a SportTester. At the commencement of exercise (Monark cycle ergometer), subjects were instructed to cycle at a standard of 70 revolutions per minute. The external work (joules) was manipulated by the experimenter according to the magnitude of the change in $T_{re}$. $T_{re}$, $f_c$, and workload adjustments were recorded every two minutes, or when a change in workload was instigated by the experimenter. Once $T_{re}$ was elevated 1°C above resting, the test was terminated and the time recorded. Subjects participated for one exposure in each of the two environmental conditions.

Subjects cycled at an intensity of 72% of age predicted maximal $f_c$ for both the neutral and heat conditions, which was sufficient to elevate $T_{re}$ within 30 minutes of the

commencement of exercise. The mean $T_{re}$ elevation was 28.4 minutes for the neutral condition and 25.4 minutes for the heat condition respectively. Since kp and rpm were recorded frequently for each subject, and the length of the flywheel of the cycle ergometer is a standard six meters, the workload in watts could be computed (Table C and D). These values were then compared to the body mass for each individual subject (watts.kg$^{-1}$) and then averaged for both the neutral and heat conditions. It was decided that the product of the mean watts.kg$^{-1}$ from this pilot study for each of the two conditions (2.1 and 1.6 watts.kg$^{-1}$ for the neutral and heat groups respectively), and the body mass of each subject participating in the acclimation regimes, would produce a sufficient initial work load to elevate $T_{re}$ within 30 minutes.

**Table C:** Estimation of the initial work load required to elevate core temperature 1°C above resting by 30 minutes following the commencement of exercise in a neutral condition: 22°C, 40% RH.

| Subject | watts | watts.kg$^{-1}$ | $f_c$ (% $f_c$ max) | $T_{re}$ elevation (mins) |
|---|---|---|---|---|
| 1 | 137.3 | 1.7 | 78 | 27 |
| 2 | 118.5 | 1.7 | 60 | 24 |
| 3 | 196.1 | 2.7 | 73 | 26 |
| 4 | 165.8 | 2.1 | 76 | 33 |
| 5 | 192.0 | 2.2 | 73 | 32 |
| mean | 161.9 | 2.1 | 72.0 | 28.4 |
| s.d. | 33.9 | 0.4 | 7.0 | 3.9 |

Abbreviations: $f_c$ = cardiac frequency; $T_{re}$ = rectal temperature

**Table D:** Estimation of the initial work load required to elevate core temperature 1°C above resting by 30 minutes following the commencement of exercise in a heat condition: 38°C, 40% RH

| Subject | watts | watts.kg$^{-1}$ | $f_c$ (% $f_c$ max) | $T_{re}$ elevation (mins) |
|---|---|---|---|---|
| 1 | 132.4 | 1.7 | 79 | 25 |
| 2 | 114.4 | 1.7 | 73 | 20 |
| 3 | 127.5 | 1.7 | 65 | 23 |
| 4 | 122.5 | 1.6 | 69 | 34 |
| 5 | 137.3 | 1.6 | 70 | 25 |
| mean | 126.8 | 1.6 | 71.2 | 25.4 |
| s.d. | 8.8 | 0.1 | 5.2 | 5.2 |

Abbreviations: $f_c$= cardiac frequency; $T_{re}$= rectal temperature

Appendix D

Thermal sensation scale

# 13-POINT
# THERMAL SENSATION SCALE

1 Unbearably cold
2 Extremely cold
3 Very cold
4 Cold
5 Cool
6 Slightly cool
7 Neutral
8 Slightly warm
9 Warm
10 Hot
11 Very hot
12 Extremely hot
13 Unbearably hot

Modified after: Gagge, A.P., Stolwijk, A.J., & Hardy, J.D. 1967. Comfort and thermal sensations and associated physiological responses at various ambient temperatures. *Environmental Research* 1:1-20.

Appendix E

Perceived exertion scale

# 15-POINT BORG SCALE

6
7 Very, very light
8
9 Very light
10
11 Fairly light
12
13 Somewhat hard
14
15 Hard
16
17 Very hard
18
19 Very, very hard
20

Borg, G.A.V. 1962. Perceived exertion in relation to physical work load and pulse rate. *Kungliga Fysioga Sallsk Lund Forh* 31:105-115.

## Appendix F

**Validation of the core temperature measurement used to determine a 1°C core temperature elevation from resting during acclimation sessions in hot and neutral environments.**

Since the NEUT group had to perform significantly more work (cycling) than the HEAT group to achieve the desired elevation in core temperature, which may induce a proportionately greater increase in $T_{re}$ than hypothalamic temperature, this pilot study was conducted post experimentally to assess the validity of using rectal temperature to determine the 1°C elevation in core temperature during acclimation sessions in both hot and neutral environments.

Four subjects participated in trials in both the neutral (22°C, RH 40%) and heat (38°C, RH 40%) environments used during acclimation. Core temperature was recorded manually from the auditory canal (aural) and the rectum. $T_{ac}$ was considered as the temperature approximately 1 cm in to the auditory canal, measured using a servo-heated aural thermometer (London Hospital Medical College, Zero-gradient aural thermometer, London). The thermometer monitors the temperature of the aural canal and the outer ear separately, both of which are insulated by a padded headset. A servo-heating circuit warms and maintains the outer ear at the temperature of the aural canal. This system provides a reliable indication of body $T_c$ (Keatinge and Sloan, 1975). $T_{re}$ was measured using a thermistor (Yellow Springs Instrument Co., Inc., YSI probe no. 401, Ohio), positioned 12 cm beyond the anal sphincter. Data was recorded manually during acclimation from a tele-thermometer (Yellow Springs Instrument Co., Inc., YSI model 46, Ohio). At the commencement of exercise (Monark cycle ergometer), subjects were instructed to cycle at a workrate established by the experimenter. This workrate was based on the pilot study explained in Appendix C. The external work (joules) was manipulated by the experimenter according to the magnitude of the change in $T_{re}$. The time, $T_{re}$ and $T_{ac}$ were recorded every two minutes, or when a change in workload was instigated by the experimenter. Once $T_{re}$ was elevated 1°C above resting,

the test was terminated and the time recorded. Subjects participated for one exposure in each of the two environmental conditions.

Analyses of the data revealed $T_{re}$ to be a valid indicator of core temperature when attempting to produce conditions of isothermal strain. An independent t-test conducted on the values attained when $T_{ac}$ was subtracted from $T_{re}$ for each time recording for each subject revealed no significant difference ($P>0.05$) between the HEAT and NEUT trials. The sensitivity of each subjects' response in each condition was plotted and regression analysis applied to determine the slope of the line. When the slopes for both core temperature measurements were compared in each of the two conditions no significant differences were found ($P>0.05$). On the basis of these results it is shown that $T_{re}$ provided a valid indication of core temperature in the attempt to achieve isothermal strain between two different thermal environments.